

# The Psychobiology of Pain Management



A Programmed  
Introduction

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# Welcome!

- I am very happy to have this opportunity to speak with you!!
- During the presentation we will discuss three questions:
  - What is pain?
  - What is hypnosis?
  - How can hypnosis be utilized to relieve pain?
- To start things off, please take a minute and write down your own answers to these three questions.

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We will return to your initial answers to these questions later on during the discussion portions of the presentation. For now, just put them aside as we continue...

# Pre-Frame

- Before we continue with the body of the presentation, I would like to share two exercises with you:
  - A quick introduction to hypnosis and trancework that will help you to access your learning abilities more easily and comfortably.
  - An overview of the basic structure of the presentation so you will be able to follow along automatically and effortlessly.

# Accessing Your Learning State

An Exercise In  
Eyes-Open Trance

# Section 1

## The Psychophysiology of Pain Perception



# 4-MAT Frame for Section 1

- What Frame: We will learn about:
  - Extremes of Pain Perception
  - Psychological Differences in Pain Perception
  - Psychological Variables in Pain Perception
  - Psychogenic Pain
  - The Language of Pain

# Extremes of Pain Perception: I

- Congenital Insensitivity to Pain
  - As children sustain:
    - Extensive burns
    - Bruises
    - Lacerations
  - Fail to respond to ruptured appendixes
  - Walk on a fractured leg until it broke completely
  - Sometimes pull out their own teeth
  - Or push their eyeballs out of their sockets

# Extremes of Pain Perception: II

- Spontaneous Pain is pain suffered by people in the apparent absence of any apparent stimulation
- Usually caused by damage to peripheral nerves from gunshot wounds or other injuries.

# Extremes of Pain Perception: III

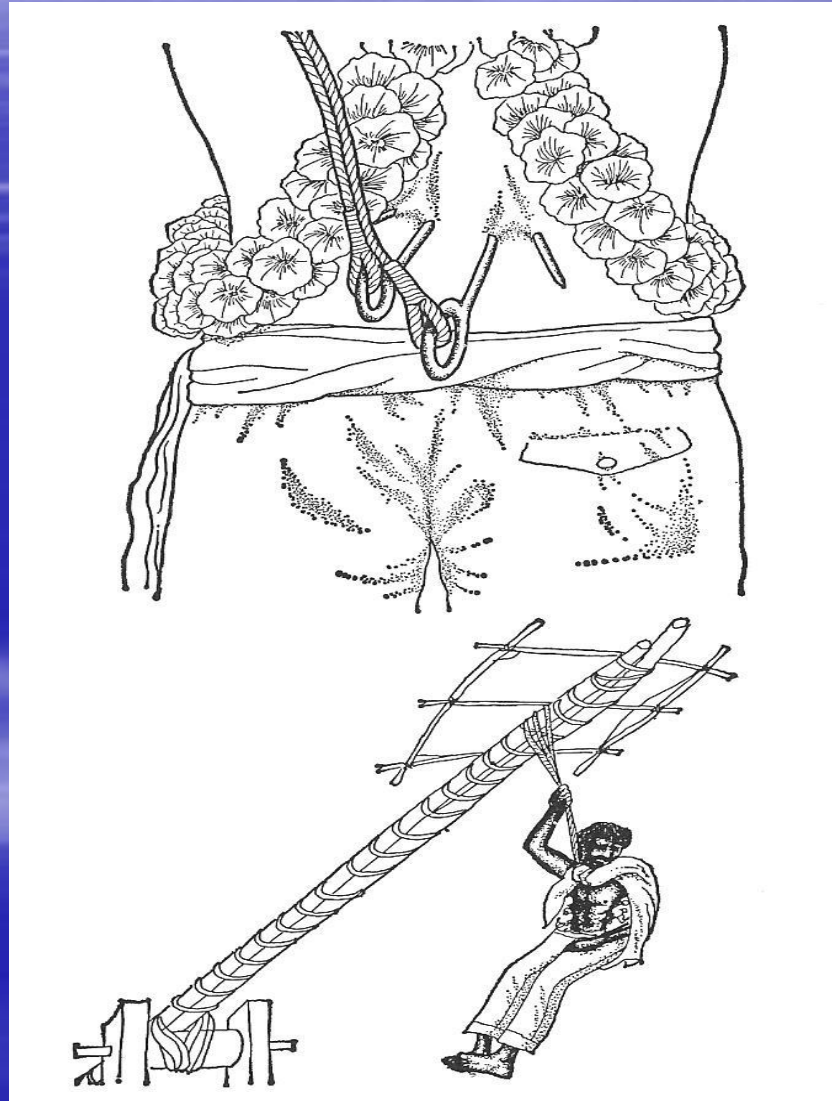
- They are described as:
  - Burning
  - Cramping
  - Shooting
- Triggered by:
  - Gentle touch
  - Puffs of air
  - No apparent stimulus



# Psychological Differences in Pain Perception

- Person to Person
- Intrapersonal
  - Same person, different times and situations
- Different Cultures
  - Childbirth [*Couvade*]
  - Hook Swinging Ritual of Eastern India
  - Sun Dance Ritual of North American Plains Indians

# East Indian Hook-Swinging



# Psychophysical Studies of 'Pain Thresholds'

- Uniform Sensation Threshold
  - The lowest stimulus value at which a sensation is first reported (Sternbach & Tursky, 1965)
- Pain Perception Threshold
  - The lowest stimulus level at which a person reports feeling pain
- Pain Tolerance Levels
  - Culturally determined



# Psychological Variables in Pain Perception: I

- Past Experience

- Melzack and Scott (1957) raised Scottish Terriers in isolation from their littermates
- They were deprived of normal stimulation, including the normal knocks and scrapes of growing up
- They would permit the experimenters to repeatedly touch their noses with a lit match or prick their skin with a pin, while their littermates would only permit these activities once.



# Psychological Variables in Pain Perception: II

- Meaning of the Situation - Beecher (1959)
  - Soldiers in combat during WWII would report feeling no pain after major battles, even though they were seriously injured
  - When asked why, they reported that they were just so happy to be alive that they felt elated, rather than in pain
  - They still responded to injections or blood drawings with feelings of pain.
  - Contrasted with civilians undergoing surgery, who universally reported feeling pain and requested increased doses of morphine

# Psychological Variables in Pain Perception: III

- Attention

- Boxers, football players, racquetball players, athletes in general can play after injury, sometimes not even recognizing that they are injured because their attention is focused so closely on the activity of their sport

- Anxiety

- Anxiety, on the other hand, increases the feelings of pain

# Psychological Variables in Pain Perception: IV

- Suggestion (Hypnosis)
  - Placebo Effect
  - 35% of those given placebos report a marked reduction in experienced pain
  - Morphine, even in large doses, will only relieve severe pain in 75% of patients
  - Studies indicate that about  $\frac{1}{2}$  of EVERY drug's effectiveness is due to placebo effects



# Psychological Variables in Pain Perception: V

## BOX 1 Comparing Efficiency of Placebo and Analgesics

Illustration of Calculation of Index of Drug Efficiency for Evaluating Placebo Efficiency Compared to Analgesic Drugs

*Index of analgesic drug efficiency:*

$$\frac{\text{Reduction in pain with unknown drug}}{\text{Reduction in pain with known analgesic (typically morphine)}}$$

*Pain criterion:*

Reduction in pain by 50% of initial level over drug level.  
or  
change in pain of 50% on rating scale (typically 10- or 5-point)

*Index of placebo efficiency for morphine:*  
(averaged across six double-blind non-crossover-design studies)

$$\frac{\text{Reduction in pain with placebo}}{\text{Reduction in pain with morphine}} = 56\%$$

Index of Placebo Efficiency Comparing Placebo with Morphine, Aspirin, Darvon, and Zomax (Derived from Available Single-Trial Double-Blind Published Studies)

Number of double-blind studies	Placebo efficiency for	%
6	Morphine	56
9	Aspirin	54
2	Darvon	54
2	Codeine	56
3	Zomax	55

Used by permission from Evans (1985).



# Psychogenic Pain: I

- Psychosomatic Pain
  - Pain that is caused by psychological processes and generally has no definable physical cause, such as lesions, injuries, or neuropathy
- Ideo-Dynamic Processes
  - Cognitive Dissonance
- Trigger Points

# Psychogenic Pain: II

Demonstration of Trigger Points

# The Language of Pain

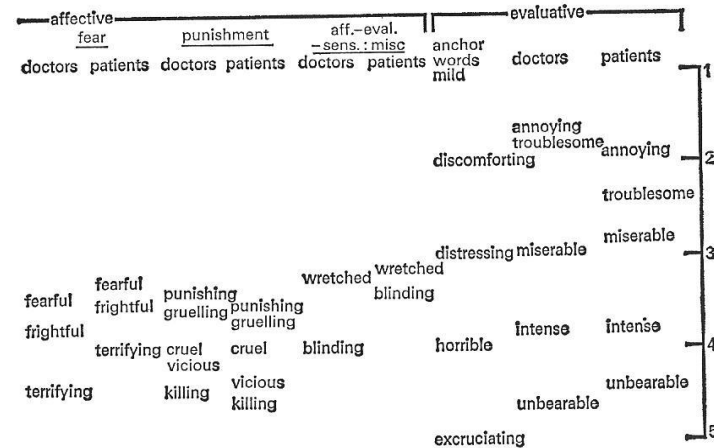
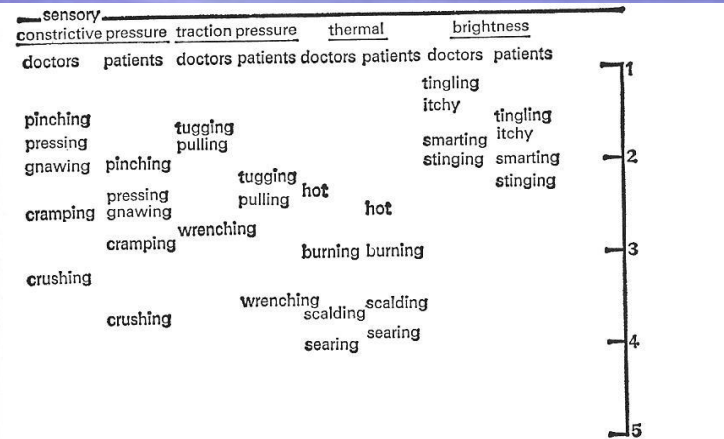
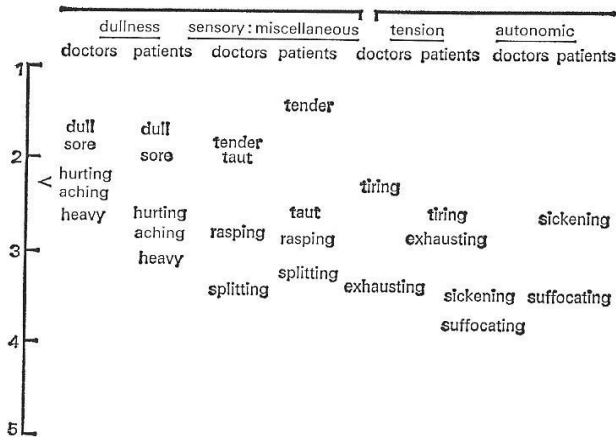
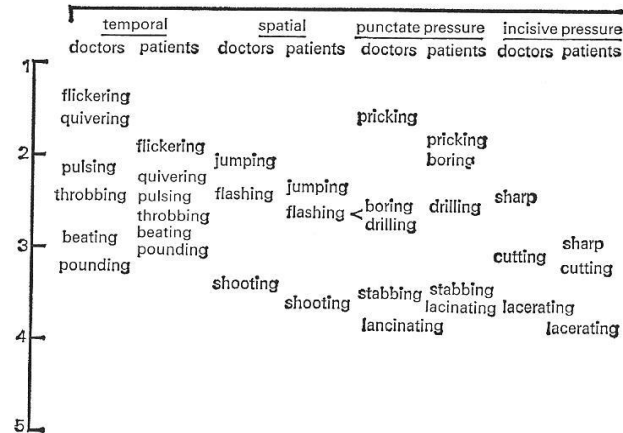


Figure 3 Spatial display of pain descriptors which have the same rank order, on an intensity scale, for doctors and patients. The scale values range from 1 (mild) to 5 (excruciating). Two words connected by an arrowhead have the same mean scale value. (from Melzack and Torgerson, 1971, p. 50)

# Tentative Definition of Pain

- Exercise: In small discussion groups, using the information presented so far [including your initial definitions of pain], design a tentative definition of pain.
- Take about 10 minutes for your discussion.
- Write down your definition.
- Select a spokesperson for your group.
- Share your definition with the group when called upon.



# Discussion Period - Questions and Answers

# Section 2

## Introduction to Your Unconscious Mind

# Introduction to Your Unconscious Mind: I

- Write your signature as you usually do at the top of the page.
- Right below that signature, write your signature slowly, as if you were in the 3<sup>rd</sup> grade learning cursive writing for the first time and practicing with your name.

# Introduction to Your Unconscious Mind: II

- Right below that line, write your signature with your non-dominant hand as carefully and slowly as you can – your left hand if you are right-handed, your right hand if you are left-handed.
- Below that line, write your signature with your non-dominant hand as quickly as you can.



# Introduction to Your Unconscious Mind: III

- Line 1: Normal Signature
- Line 2: Slow Signature, Dominant Hand
- Line 3: Slow Signature, Non-Dominant Hand
- Line 4: Fast Signature, Non-Dominant Hand

# Analysis of the Handwriting Exercise

Signature Sample	Conscious or Unconscious	Competency Level
1: Dominant hand, normal signature	Unconscious: free-flowing, no deliberation, rapid, unaware of the movements of the hand, wrist, fingers.	Unconscious competence – high level competence requiring no conscious direction or deliberation.
2. Dominant hand, careful signature	Conscious: deliberate, slow, careful. You are fully aware of each movement.	Conscious competence – next level competence directed by conscious mind.



Signature Sample	Conscious or Unconscious	Competency Level
3. Non-dominant hand, careful signature	Conscious: deliberate, slow, extremely careful – you are even more fully aware of the movements and sensations in your non-dominant hand.	Conscious incompetence – the conscious mind doesn't know how to perform this task yet, so it is slow-going and awkward. Practice would make it easier.
4. Non-dominant hand, rapid signature	Unconscious: free-flowing, but sloppy, little awareness of the sensations in the hand except for a feeling of awkwardness.	Unconscious incompetence – not even the unconscious mind has developed the skills to perform this task yet. Practice for a few weeks would lead to unconscious competence and signatures with either hand would be very good.



# Discussion Period - Questions and Answers

# Section 3

## The Clinical Puzzle of Pain

# 4-MAT Frame for Section 3

- What Frame: This section presents information on 4 kinds of perplexing pain syndromes:
  - Phantom Limb Pain
  - Causalgia
  - The Neuralgias
  - Post Traumatic Pain

# Phantom Limb Pain: I

- Phantom Limb Pain is pain that is localized in the area of an amputated limb.
- The pain endures long after healing of the amputated limb.
  - In about 70% of the cases, the pain persists for a year or longer.
  - In many cases, it can persist for years or even decades.



# Phantom Limb Pain: II

- Trigger zones may spread to healthy areas on the ipsilateral or contralateral sides of the body.
  - These trigger zones, when stimulated with mild pressure or other stimuli, will generate sensations of pain in the amputated limb.
  - There are many amputees who suffer phantom limb pain during episodes of angina pectoris.

# Phantom Limb Pain: III

- Phantom limb pain is more likely to develop in patients who have suffered pain in the limb for some time before the amputation.
- Treatment for phantom limb pain is usually only temporary, and may involve:
  - Injections of anesthetic at the site of the amputation
  - Injections of saline solution
  - Vigorous vibration at the site
  - Hypnotherapy
  - Cognitive-behavioral therapy
  - Surgical interventions

# Phantom Limb Pain: IV

- Causes of phantom limb pain:
  - Peripheral nerve mechanisms
    - Low level stimuli of the skin or muscles
  - Sympathetic nervous system mechanisms
    - Poor blood flow
    - Coldness
    - Sweating

# Phantom Limb Pain: V

- Causes of phantom limb pain (cont.):
  - Psychological mechanisms
    - Emotional disturbances
    - Anxiety
    - Psychopathology



# Causalgia: I

- Causalgia is a severe burning pain associated with a rapid, violent deformation of nerves by high-velocity missiles, such as bullets or shrapnel.
- It occurs in 2-5% of cases of peripheral nerve injury, and is typically seen in young men that have been injured in combat.
- It usually persists for up to 6 months, and in 25% of the cases, for 12 months or more.

# Causalgia: II

- Like phantom limb pain, causalgia is triggered by normally non-noxious stimuli such as touch or light pressure.
- Treatment for causalgia is the same as for phantom limb pain:
  - Injections
  - Hypnotherapy and Cognitive-Behavioral Therapy
  - Surgery

# Causalgia: III

- Sympathetic nervous system inputs seem to play a large role in the pain of causalgia:
  - The area is cold
  - Drips sweat
  - Is discolored
  - On the hand, even the fingernails become brittle and shiny



# The Neuralgias: I

- There are several pain syndromes associated with peripheral nerve damage that are generally categorized as neuralgic pain.
- The properties of the neuralgias are similar to phantom limb pain and causalgia:
  - Unremitting pain which is
  - Difficult to treat



# The Neuralgias: II

- Neuralgias can be caused by many factors:
  - Viral nerve infections (herpes zoster)
  - Diabetic nerve degeneration
  - Poor circulation in the limbs
  - Poisons such as arsenic and lead

# Post Traumatic Pain

- Post traumatic pain occurs after various kinds of accidents.
- Post traumatic pain:
  - Persists long after the injuries have healed
  - Severity exceeds expectations
  - Pain and trigger zones may spread to other areas of the body
  - Is difficult to diagnose and treat

# Implications of the Clinical Evidence

- There are 7 implications of the evidence from these pathological pain syndromes that help to guide our understanding of what pain is and how it can be successfully treated.
  1. Summation of peripheral nerve impulses
  2. Multiple contributions to the sensation of pain
  3. Delays in the perception of pain after a stimulus is applied
  4. Persistence of the pain long after the stimulus
  5. Spread of the pain to non-affected areas
  6. Resistance to surgical control
  7. Relief by modulation of the sensory input

# Discussion Period - Questions and Answers



# Section 4

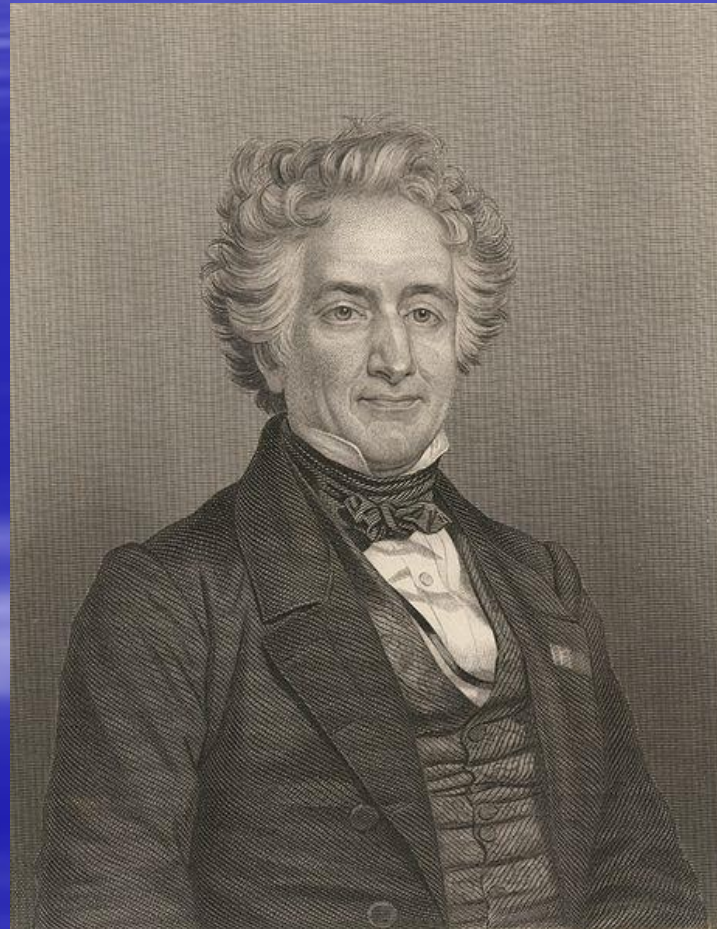
## Communicating With Your Unconscious Mind

# 4-MAT Frame for Section 4

- What Frame: This section presents information on:
  - Michel Eugene Chevreul
  - Ideo-dynamic processes
  - The Chevreul Pendulum
- This section also presents an exercise using the Chevreul Pendulum to communicate with your own Unconscious Mind

# Michel Eugene Chevreul

(1786-1889)





# Ideo-Dynamic Processes

- Ouija Boards
- Dowsing
- Automatic Writing
- Facilitated Communication
- Chevreul Pendulum
- Hypnotic Finger Signaling
- Kinesiology



# Working With The Chevreul Pendulum

# Discussion Period - Questions and Answers

# Section 5

## The Physiology of Pain

# 4-MAT Frame for Section 5

- What Frame: This section presents information on:
  - The neurophysiological structures underlying pain sensations and perceptions
- Why Frame: This information is important because it leads us systematically toward a complete theory of pain

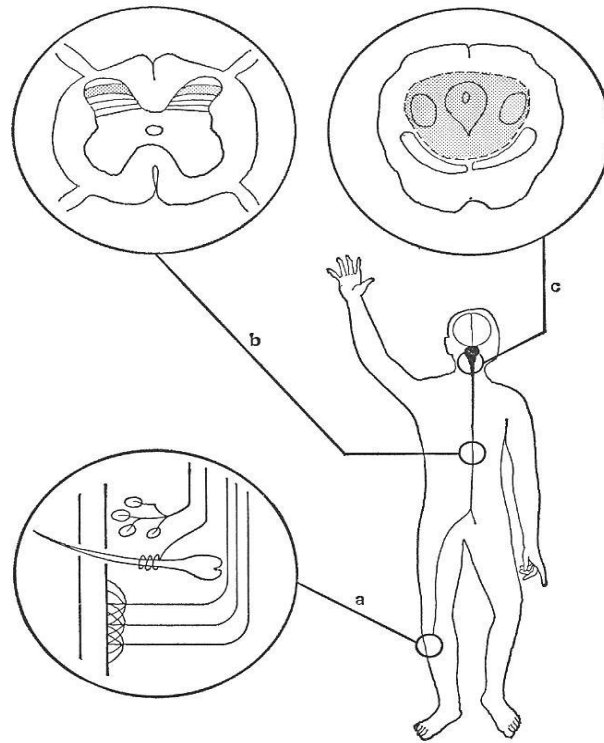


# Classical Cartesian Model



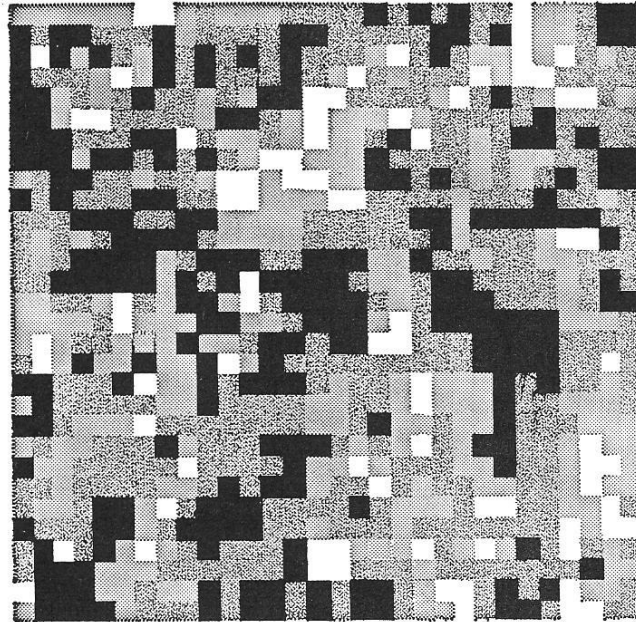
Figure 22 Descartes' (1644) concept of the pain pathway. He writes: 'If for example fire (A) comes near the foot (B), the minute particles of this fire, which as you know move with great velocity, have the power to set in motion the spot of the skin of the foot which they touch, and by this means pulling upon the delicate thread (cc) which is attached to the spot of the skin, they open up at the same instant the pore (d e) against which the delicate thread ends, just as by pulling at one end of a rope one makes to strike at the same instant a bell which hangs at the other end.'

# Somatic Receptor Pathways

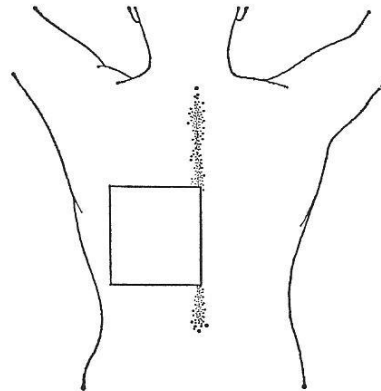


**Figure 7** Schematic representation of the receptors and projection pathways of the somatic sensory system. **A:** The diagram of the skin shows widely branching free nerve-endings (which produce overlapping receptive fields) as well as some specialized end-organs. The fibres project to the spinal cord. **B:** The cross section of the spinal cord shows the laminae (layers) of cells in the dorsal horns which receive sensory fibres and project their axons toward the brain. The cross-hatched area represents the substantia gelatinosa (laminae 2 and 3). **C:** The brainstem (lower part of the brain) receives a large somatosensory input, and projects to higher as well as lower areas of the central nervous system. The cross-hatched area represents the reticular formation. Below it on each side is the medial lemniscus. The spinothalamic projections – which are shown within the reticular formation – lie above the lemniscal tracts.

# Neural Specificity

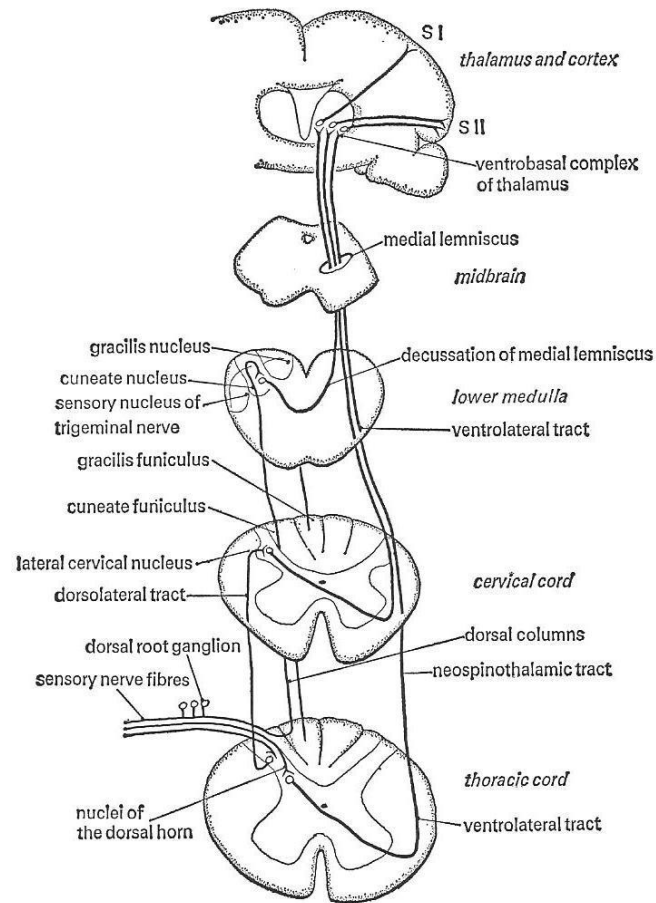


- strong cold
- ▒ moderate cold
- ░ mild cold
- touch, no cold





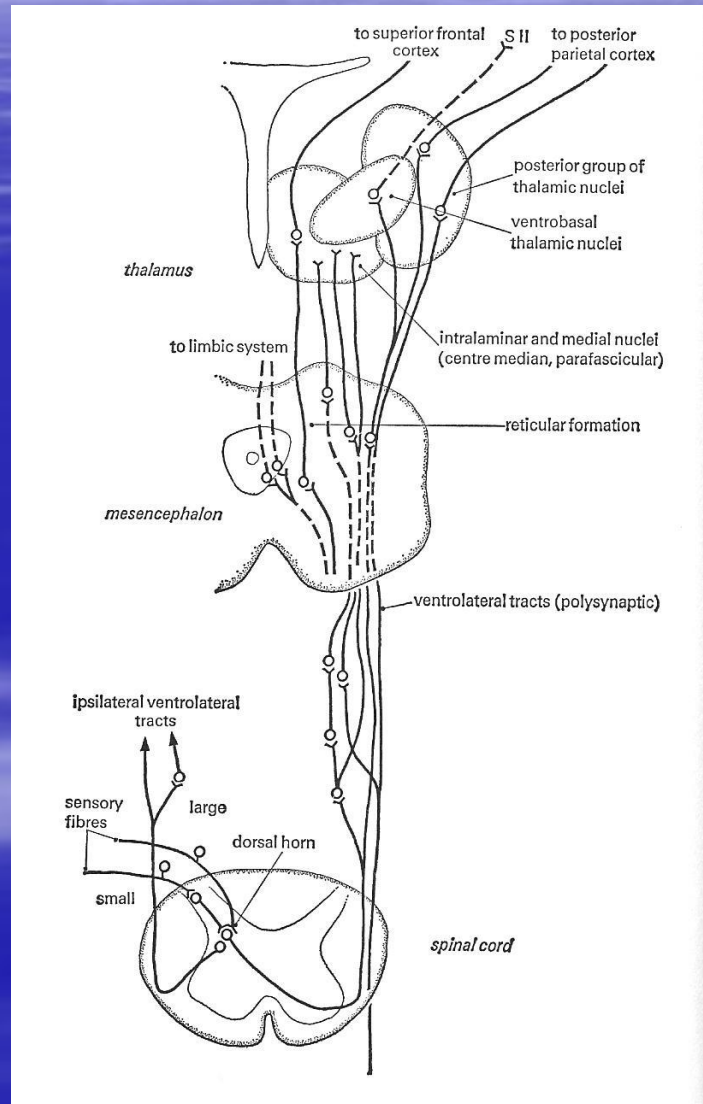
# Rapidly Conducting Fibers



**Figure 10** The rapidly conducting somatosensory projection pathways. The three main projection pathways are the dorsal column-medial lemniscal pathway, the dorsolateral tract (of Morin), and the neospinothalamic tract. The lower sections are shown on a larger scale than the upper sections. (from Milner, 1970)



# Slow Conducting Fibers



# Spatio-Temporal Patterning

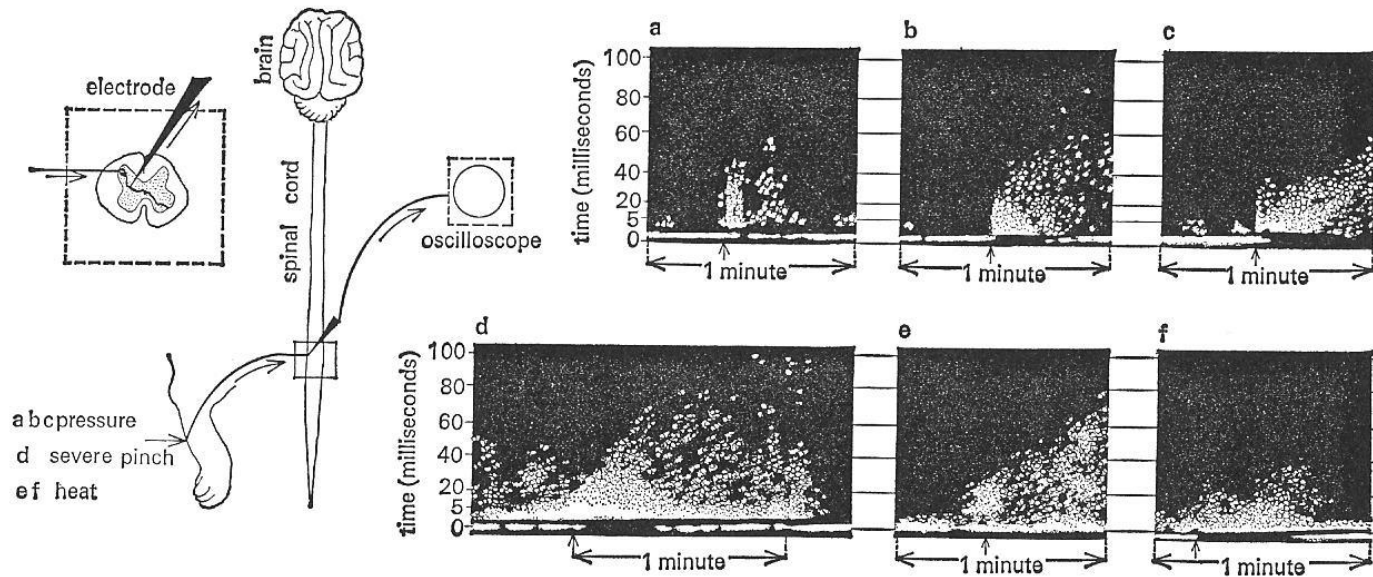
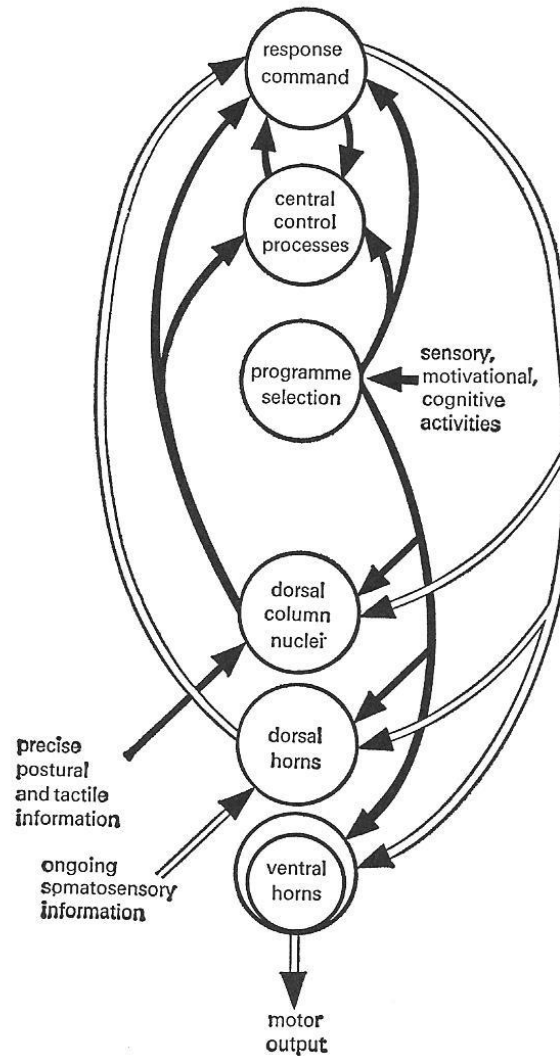


Figure 17 Neuron firing patterns, recorded from single cells in the spinal cord of a cat, show the initial response in the central nervous system to various stimuli applied to the cat's leg. Pattern a was caused by hanging a two-gram weight on a single hair; b shows the effect of a twenty-gram weight; c is the effect of a mild pinch. All three stimuli start at the arrows and continue for the duration of the recording. In d the skin was severely pinched for one minute. In e and f a heat lamp was directed at the skin for fifteen seconds after the arrows, raising the skin temperature four and twelve degrees centigrade respectively. Each dot in the recordings represents a single nerve impulse; height above base line represents time interval between recorded impulse and preceding one.  
(from Wall, 1960, and Wall and Cronly-Dillon, 1960, p. 365)

# Motor Neurons





# Discussion Period - Questions and Answers



# Section 6

## The Evolution of Pain Theories

# 4-MAT Frame for Section 6

- What Frame: This section presents information on:
  - The historical and experimental models of pain
- Why Frame: This information is important because it adds to our understanding of the way we have arrived at our current pain theories

# Classical Cartesian Model



Figure 22 Descartes' (1644) concept of the pain pathway. He writes: 'If for example fire (A) comes near the foot (B), the minute particles of this fire, which as you know move with great velocity, have the power to set in motion the spot of the skin of the foot which they touch, and by this means pulling upon the delicate thread (cc) which is attached to the spot of the skin, they open up at the same instant the pore (d e) against which the delicate thread ends, just as by pulling at one end of a rope one makes to strike at the same instant a bell which hangs at the other end.'

# Livingston's Model (1943)

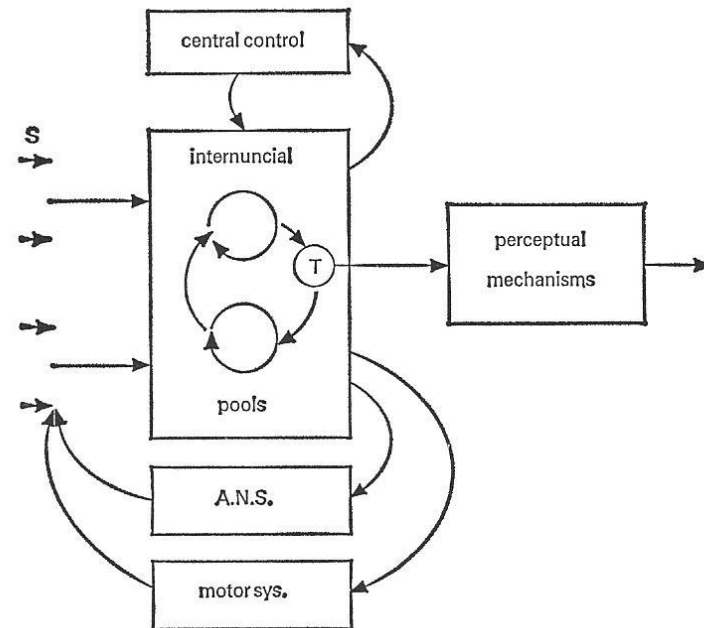


Figure 24 Schematic diagram of W. K. Livingston's (1943) theory of pathological pain states. The intense stimulation (S) resulting from nerve and tissue damage activates fibres that project to internuncial neuron pools in the spinal cord, creating abnormal reverberatory activity in closed self-exciting neuron loops. This prolonged, abnormal activity bombards the spinal cord transmission (T) cells which project to brain mechanisms that underlie pain perception. The abnormal internuncial activity also spreads to lateral and ventral horn cells in the spinal cord, activating the autonomic nervous system (A.N.S.) and motor system, producing sweating, jactitations, and other manifestations. These, in turn, produce further abnormal input, thereby creating a 'vicious circle'. Brain activities such as fear and anxiety evoked by pain also feed into and maintain the abnormal internuncial pool activity.



# Sensory Interaction Theory

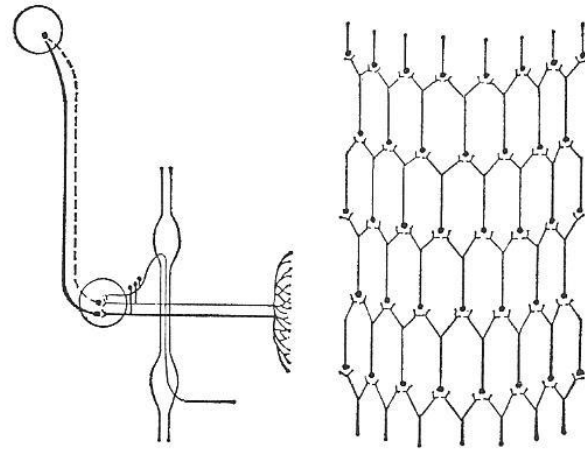


Figure 25 Noordenbos' (1959) concept of pain mechanisms. *Left:* small-diameter, slowly conducting somatic afferents and small visceral afferents (which travel through the sympathetic ganglia) project onto cells in the dorsal horn of the spinal cord. The summation of inputs from the small fibres produces the neural patterns that are transmitted to the brain to produce pain. The large-diameter fibres inhibit transmission of impulses from the small fibres and prevent summation from occurring. A selective loss of large fibres brings about a loss of inhibition and thereby increases the probability of summation and abnormal pain phenomena. The small fibre projection system – drawn as a dashed line – is indicated to be multi-synaptic. *Right:* Noordenbos' representation of the 'Multi-synaptic Afferent System' in the spinal cord. The diffuse, widespread conduction through the system, Noordenbos suggests, is the basis of 'the leak' of nerve signals that evoke pain even after extensive surgical section of the anterolateral pathways.

# Affect Theory of Pain

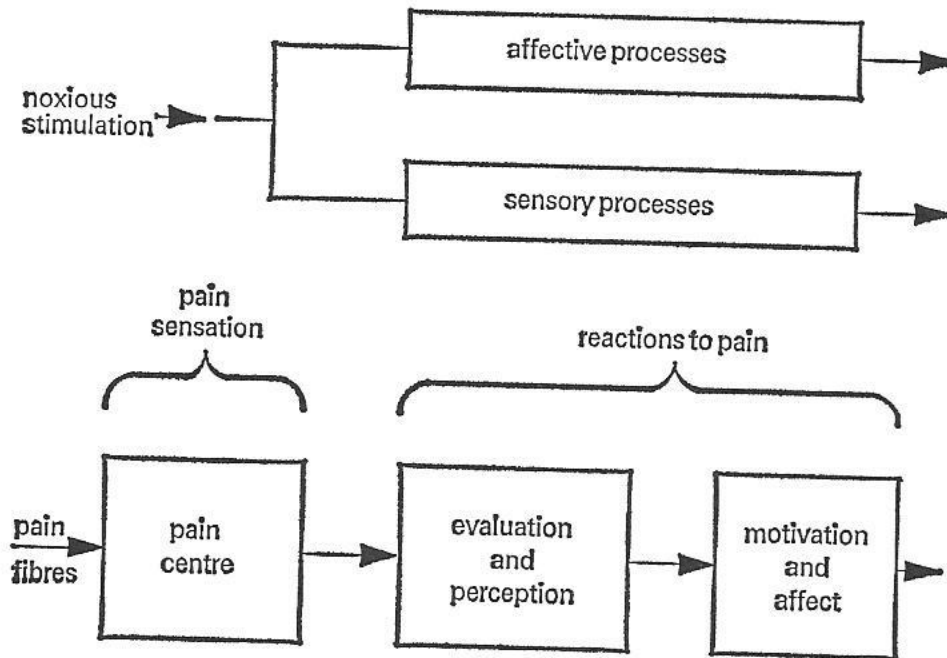


Figure 26 *Top*: diagram of Marshall's (1894) concept of pain as an affective quality or *quale*. Intense stimulation of the skin activates two parallel systems: one is the basis of the affective properties of the experience, the other underlies the sensory properties. *Bottom*: diagram of the concept, implicit in specificity theory, that motivation and affect are reactions to pain, but are not part of the primary pain sensation. (from Melzack and Casey, 1968)

# Review of Models

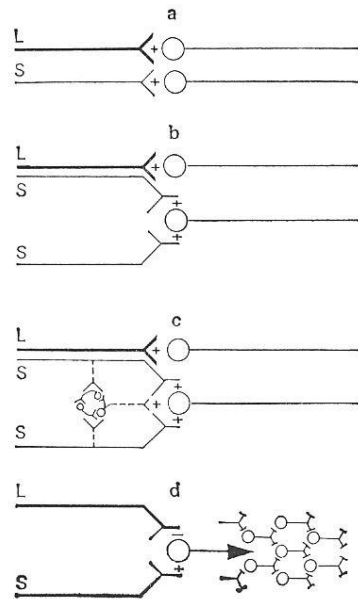


Figure 27 Schematic representation of conceptual models of pain mechanisms. A: von Frey's specificity theory. Large (L) and small (S) fibres are assumed to transmit touch and pain impulses respectively, in separate, specific, straight-through pathways to touch and pain centres in the brain. B: Goldscheider's summation theory, showing convergence of small fibres onto a dorsal horn cell. Touch is assumed to be carried by large fibres. C: Livingston's (1943) conceptual model of reverberatory circuits underlying pathological pain states. Prolonged activity in the self-exciting chain of neurons bombards the dorsal horn cell, which transmits abnormally patterned volleys of nerve impulses to the brain. D: Noordenbos' (1959) sensory interaction theory, in which large fibres inhibit (—) and small fibres excite (+) central transmission neurons. The output projects to spinal cord neurons which are conceived by Noordenbos to comprise a Multi-synaptic Afferent System.  
(from Melzack and Wall, 1970, p. 3)



# Discussion Period - Questions and Answers



# Section 7

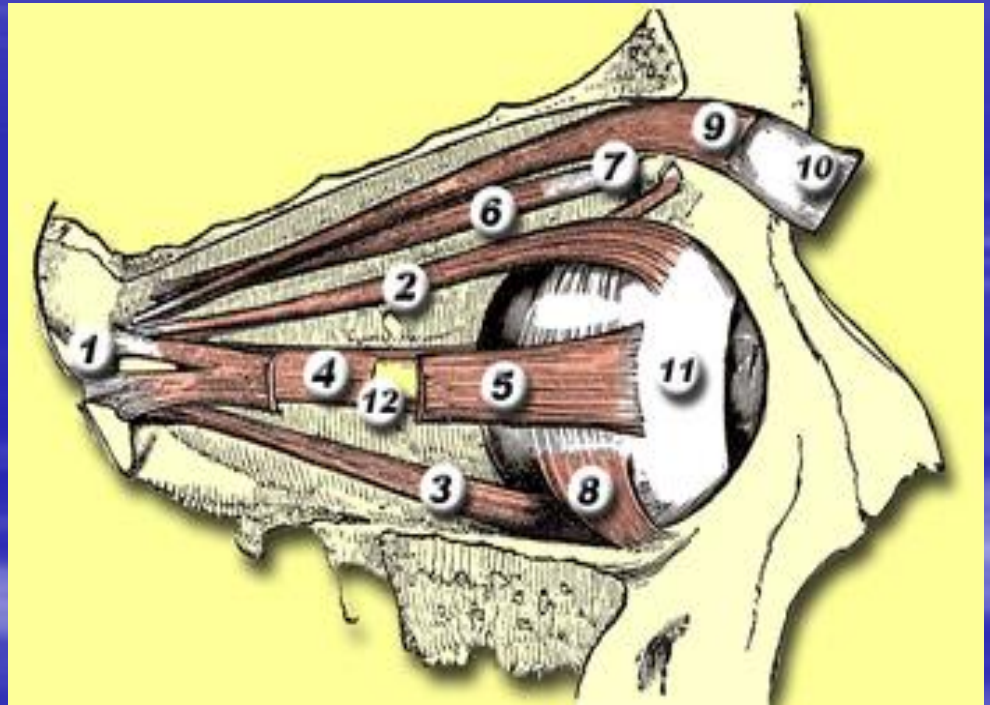
## The Relaxation Response – Part I

# 4-MAT Frame for Section 7

- What Frame: This section presents information on:
  - The anatomy of the eye and skull
  - An Eye Relaxation Exercise

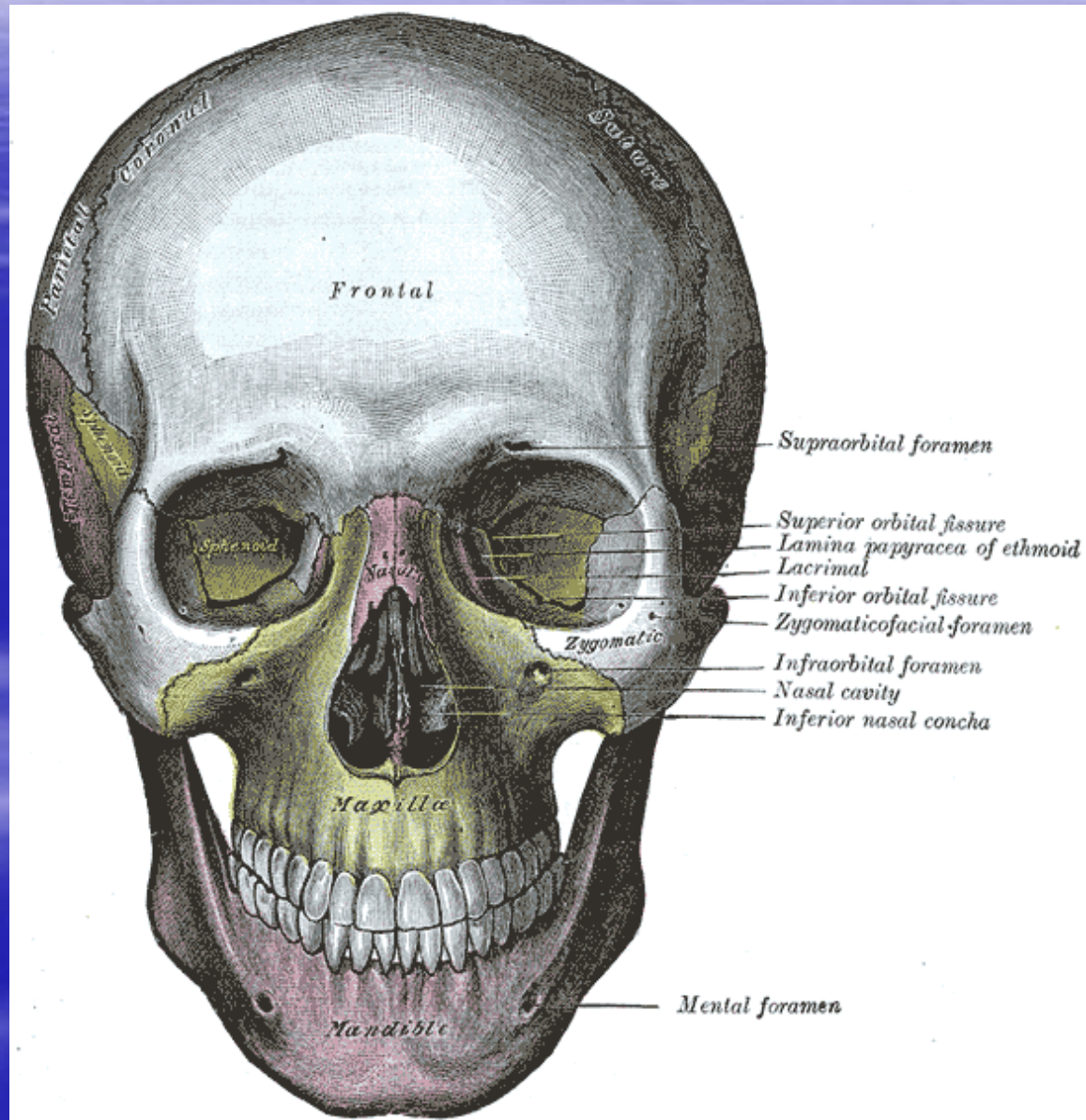
# Myostructure of the Eye

- 1 = Anulus tendineus communis
- 2 = Superior rectus muscle
- 3 = Inferior rectus muscle
- 4 = Medial rectus muscle
- 5 = Lateral rectus muscle
- 6 = Superior oblique muscle
- 7 = Trochlea
- 8 = Inferior oblique muscle
- 9 = Levator palpebrae superioris muscle
- 10 = Eyelid
- 11 = Eyeball
- 12 = Optic nerve





# Skeletal Structure of the Skull





# Eye Relaxation Exercise

# Discussion Period - Questions and Answers

# Section 8

## The Gate Control Theory of Pain

# 4-MAT Frame for Section 8

- What Frame: This section presents information on:
  - The current theory of pain, called the Gate Control Theory
  - It will tie together all of the earlier information from our historical and neurophysiological studies



# The Gate Control Theory (1965)

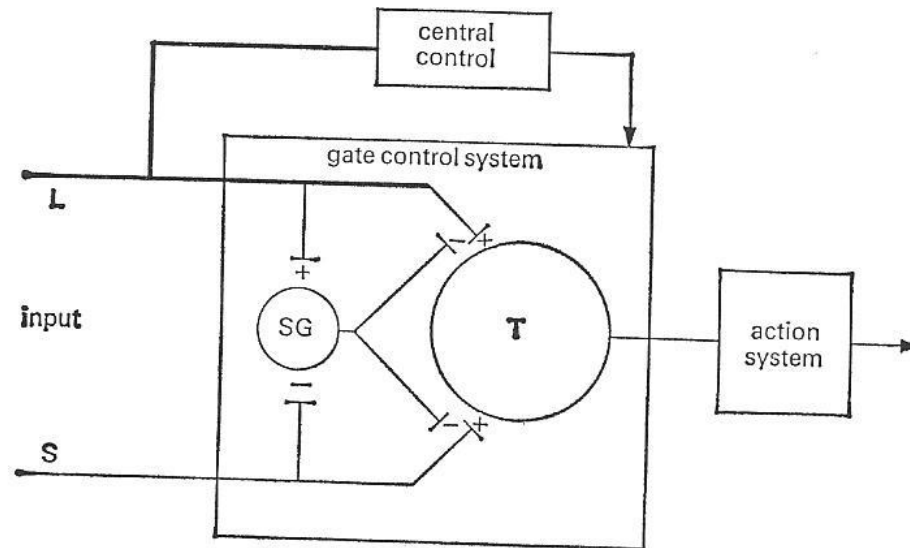
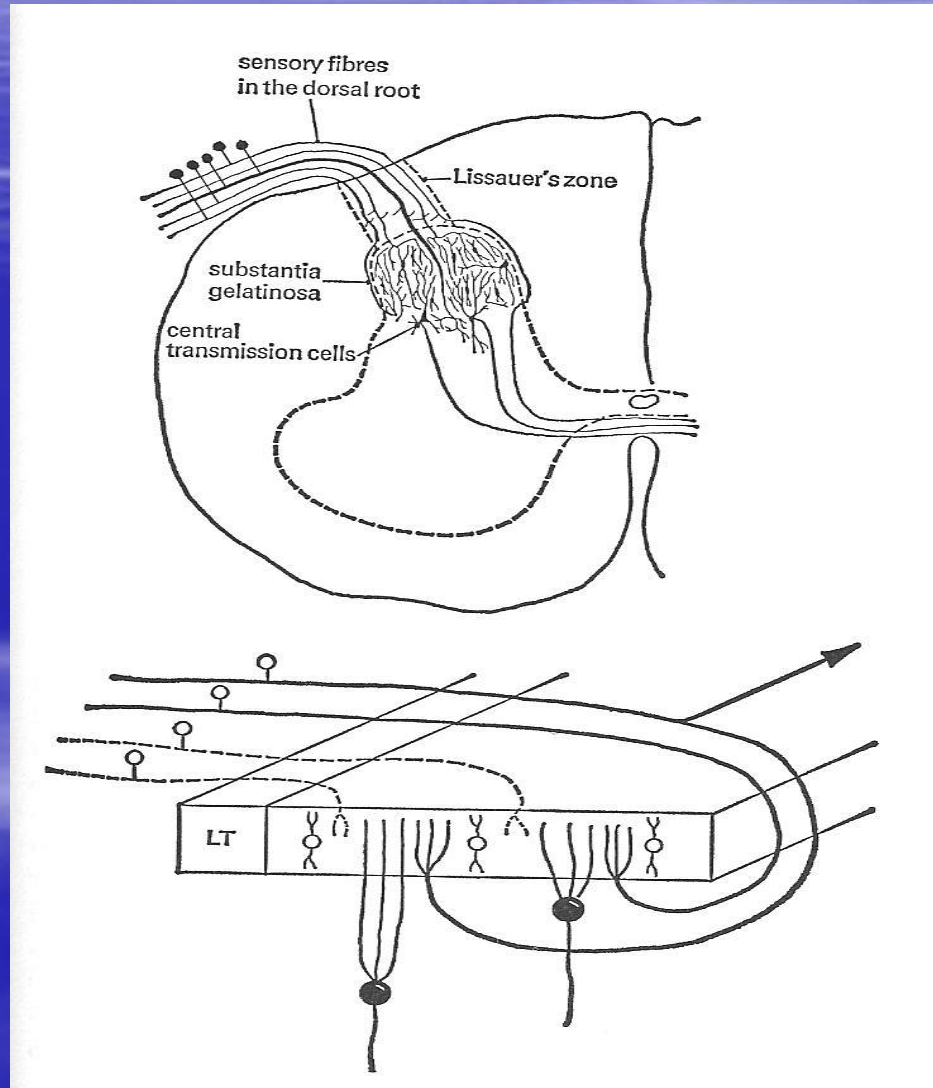


Figure 28 Schematic diagram of the gate-control theory of pain mechanisms: L, the large-diameter fibres; S, the small-diameter fibres. The fibres project to the substantia gelatinosa (SG) and first central transmission (T) cells. The inhibitory effect exerted by SG on the afferent fibre terminals is increased by activity in L fibres and decreased by activity in S fibres. The central control trigger is represented by a line running from the large fibre system to the central control mechanisms; these mechanisms, in turn, project back to the gate-control system. The T cells project to the entry cells of the action system. +, excitation; -, inhibition. (from Melzack and Wall, 1965, p. 971)

# Spinal Gating Mechanism



# Action Control System

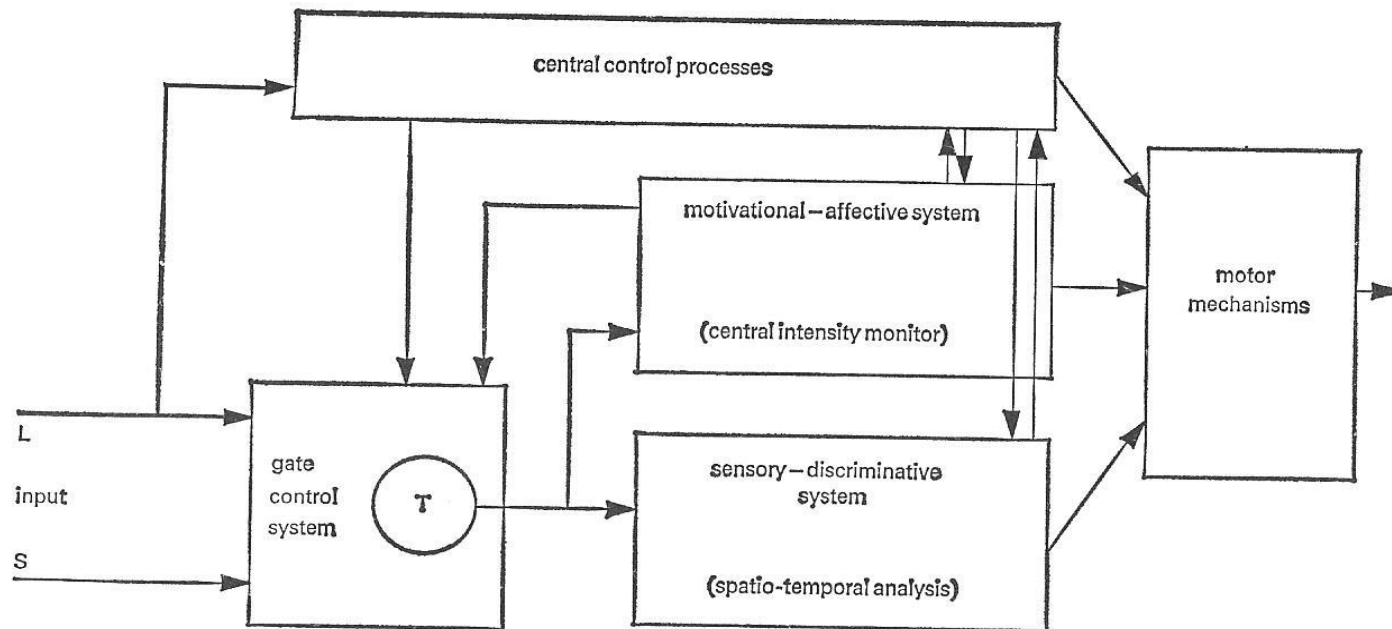
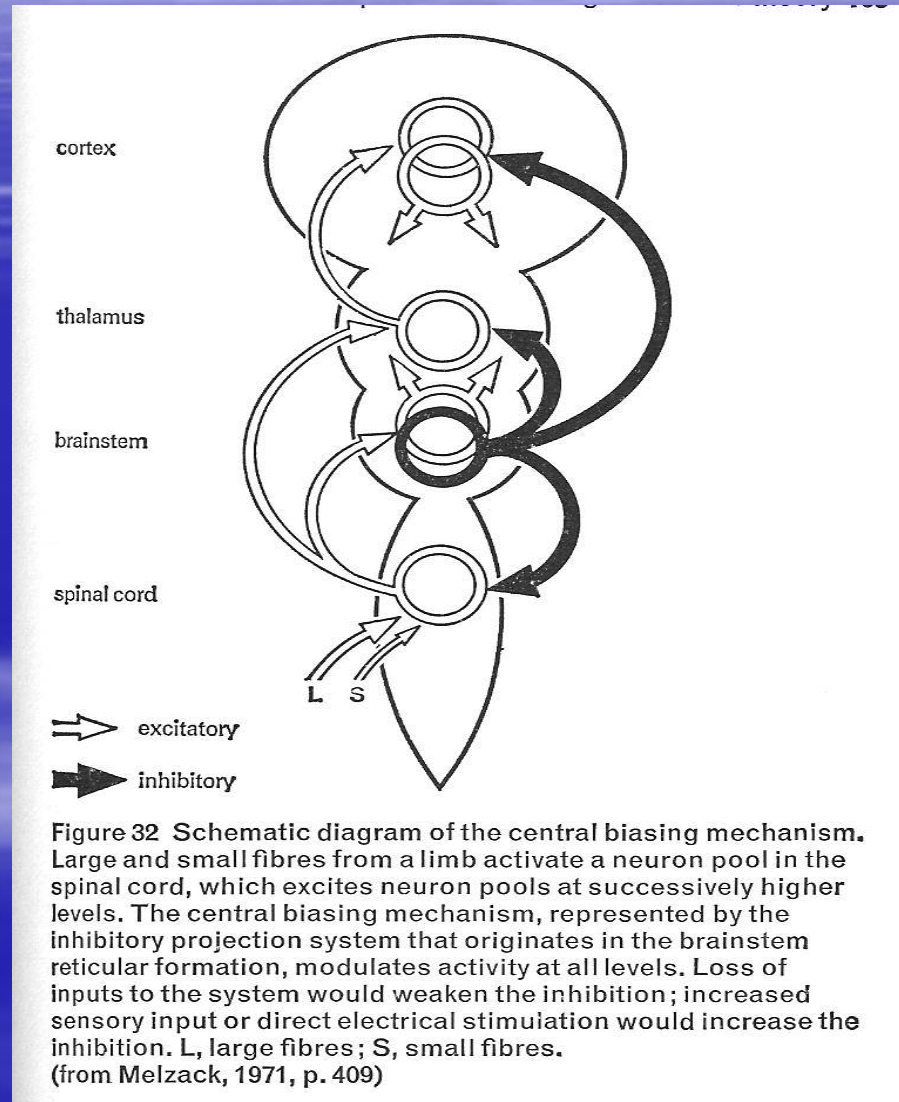


Figure 30 Conceptual model of the sensory, motivational and central control determinants of pain. The output of the T cells of the gate-control system projects to the sensory-discriminative system (via neospinothalamic fibres) and the motivational-affective system (via the paramedial ascending system). The central control trigger is represented by a line running from the large fibre system to central control processes; these, in turn, project back to the gate-control system, and to the sensory-discriminative and motivational-affective systems. All three systems interact with one another, and project to the motor system. (from Melzack and Casey, 1968)



# Central Biasing Mechanism





# Discussion Period - Questions and Answers

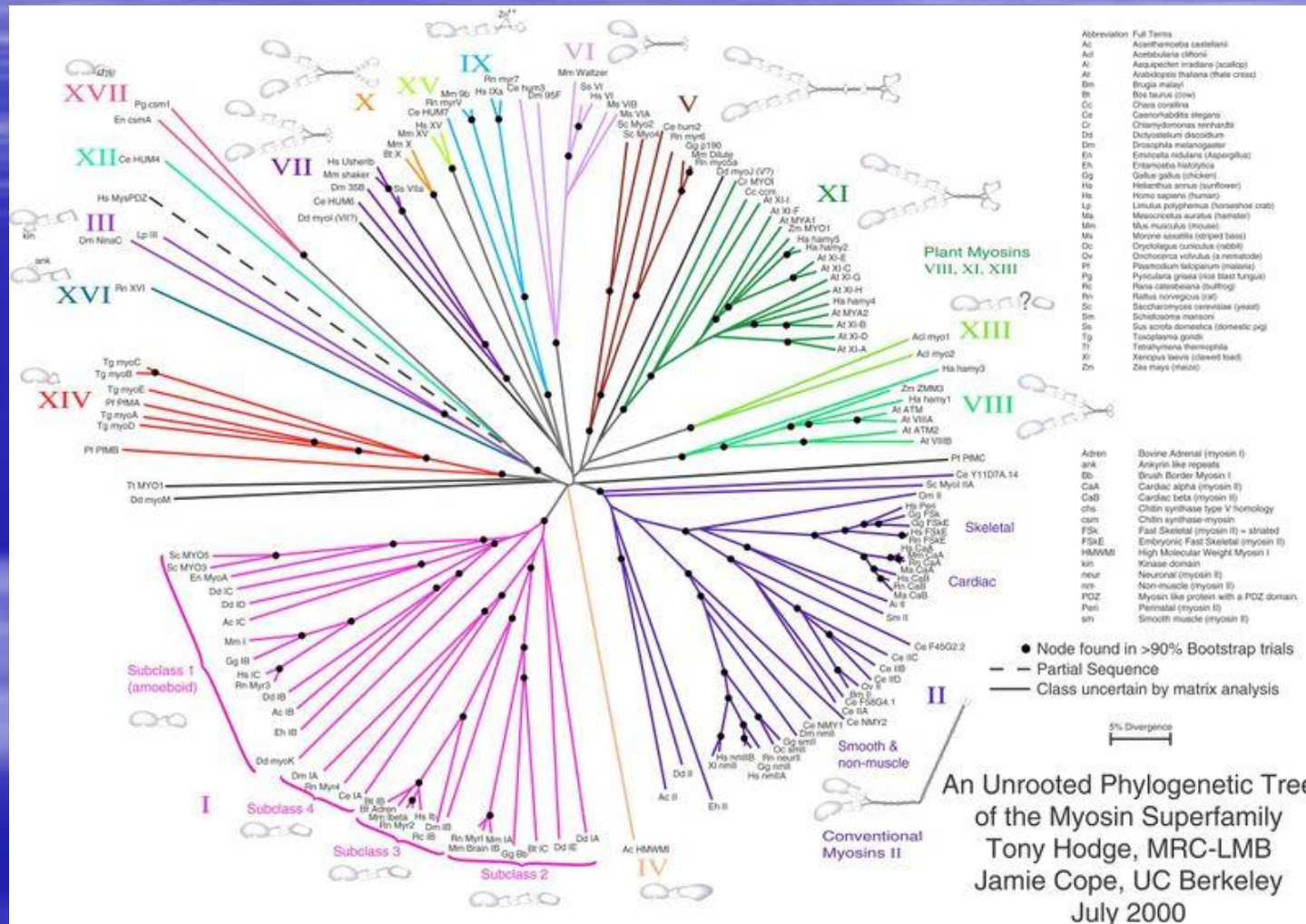
# Section 9

## The Relaxation Response – Part II

# 4-MAT Frame for Section 9

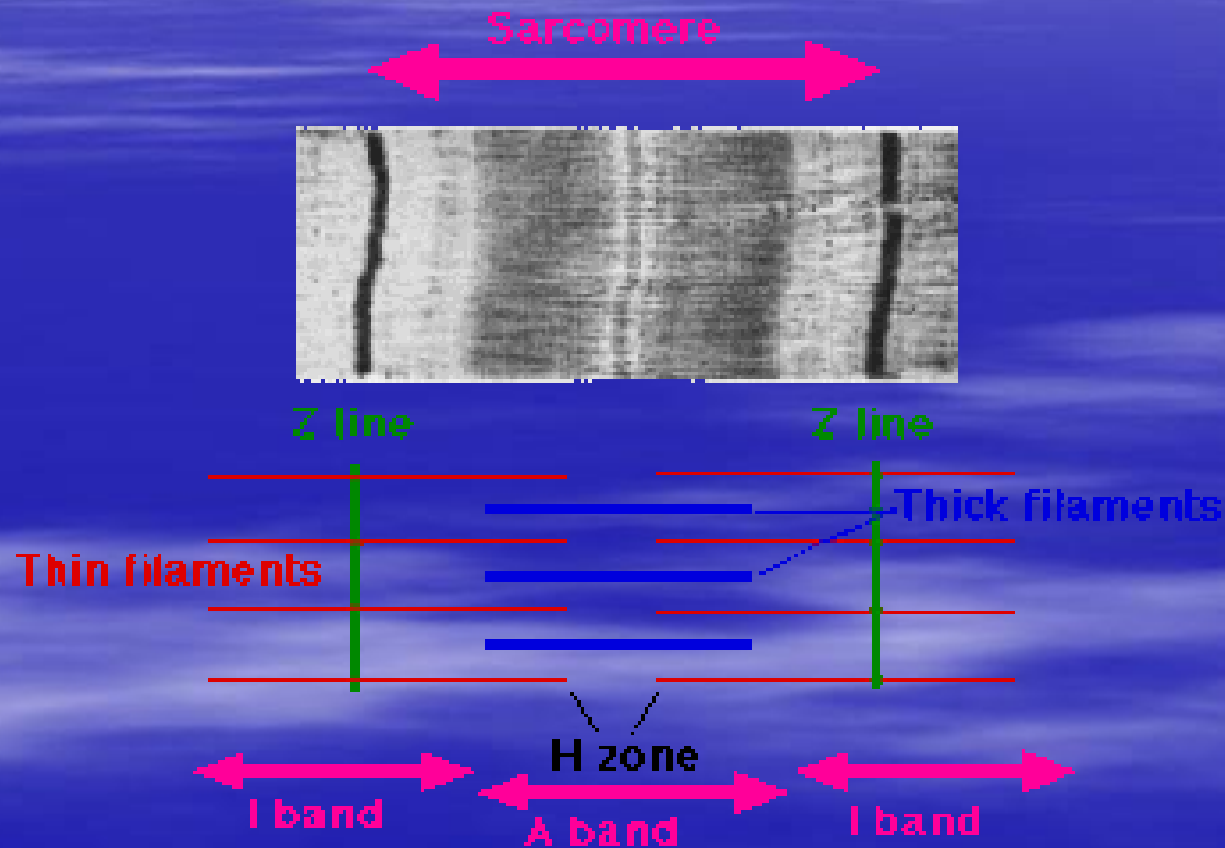
- What Frame: This section presents information on:
  - Muscular neuroanatomy
  - An exercise in relaxation called Differential Muscular Relaxation



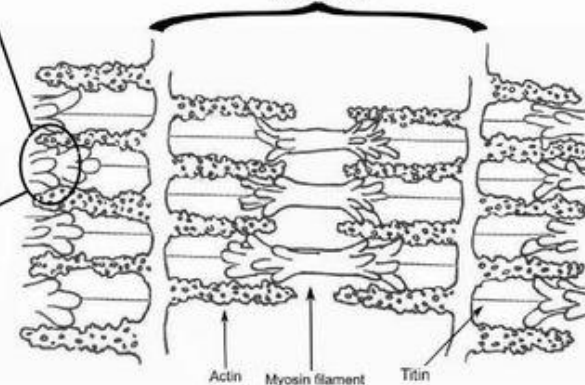
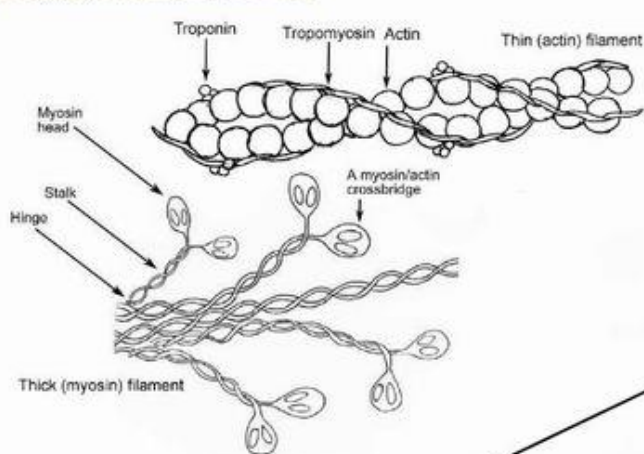
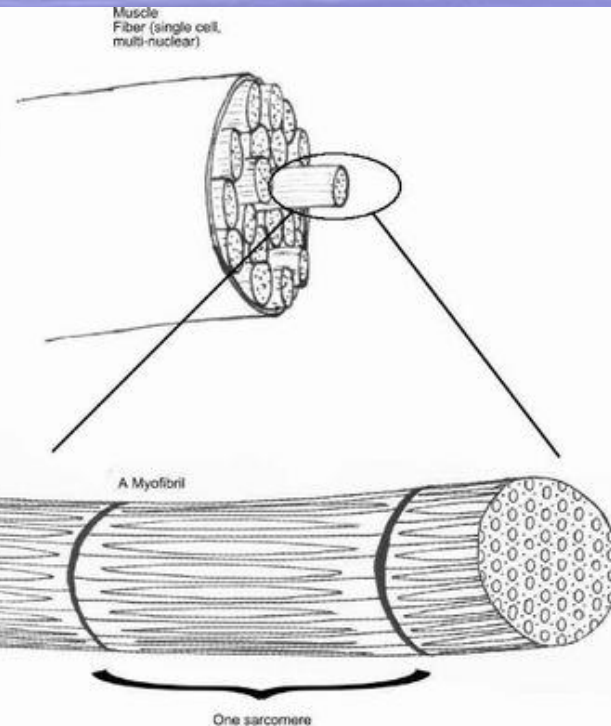
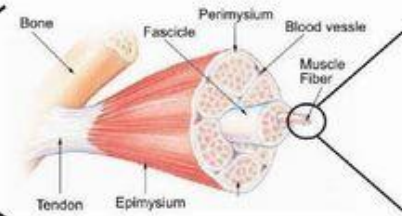




# Myo-Structure: Sarcomere



# Myo-Structures



# Differential Muscular Relaxation Exercise

# Discussion Period - Questions and Answers



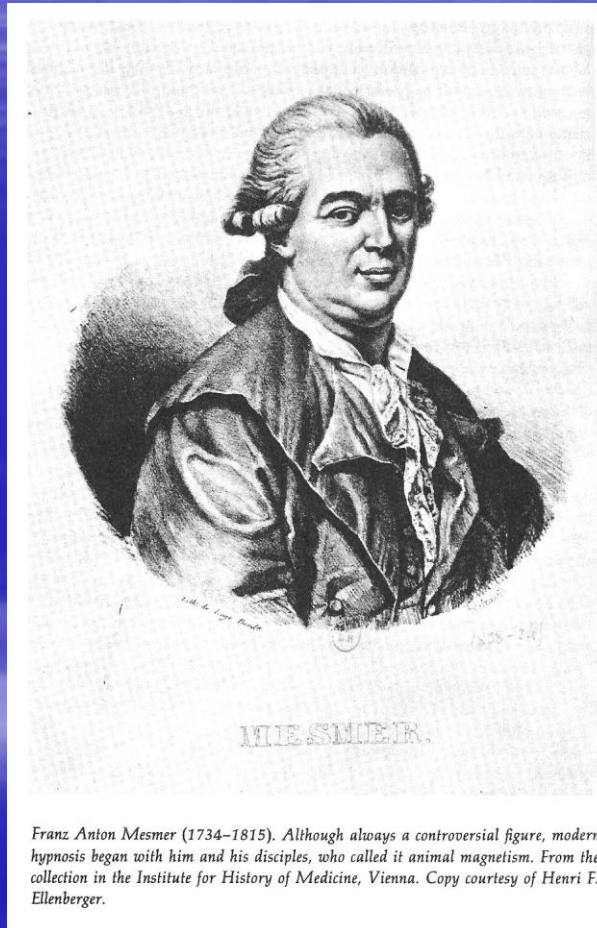
# Section 10

## Introduction to Hypnosis

# 4-MAT Frame for Section 10

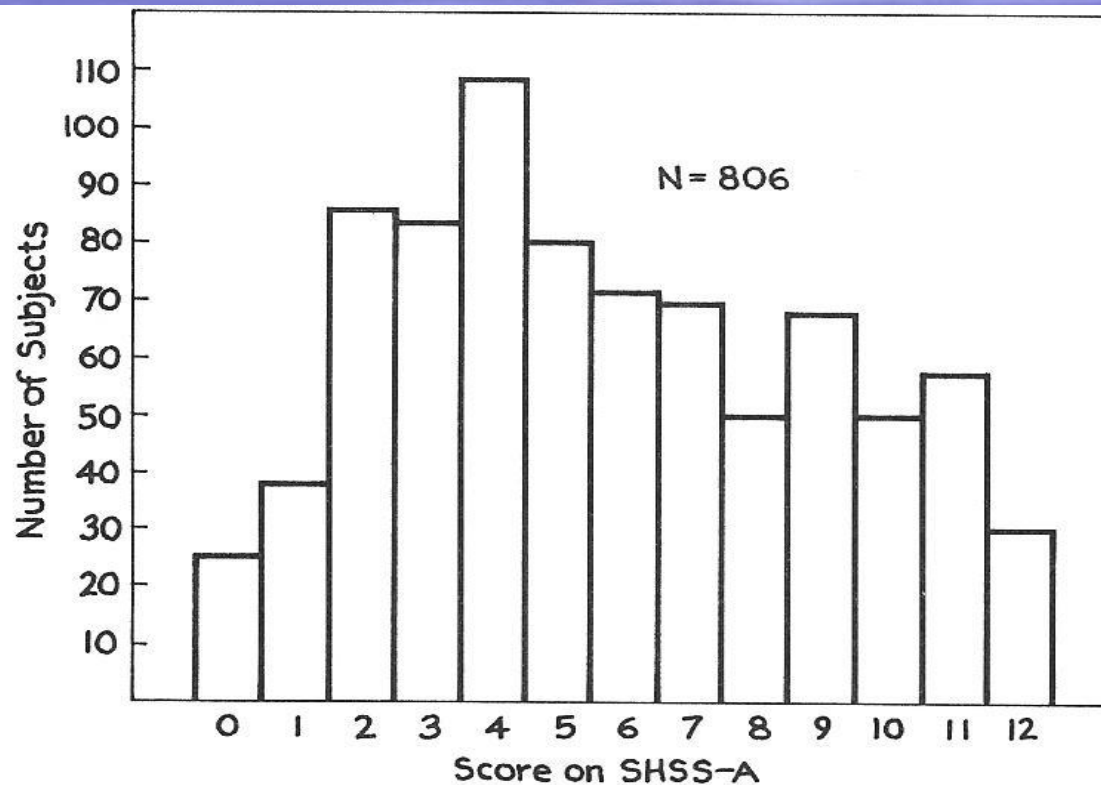
- What Frame: This section presents information on:
  - The origins of hypnosis
  - An EEG-State definition of hypnosis
  - The neuroscience behind hypnotic effects
  - Ultradian Rhythms and their effects on our health and well-being
  - Gene Expression as the basis for hypnotic effects

# Franz Anton Mesmer



*Franz Anton Mesmer (1734–1815). Although always a controversial figure, modern hypnosis began with him and his disciples, who called it animal magnetism. From the collection in the Institute for History of Medicine, Vienna. Copy courtesy of Henri F. Ellenberger.*

# Hypnotic Responsiveness



**Figure 1.** Hypnotic responsiveness scores of 806 college students. The scores were earned on individual tests with the Stanford Hypnotic Susceptibility Scale, Form A; the least responsive scored 0, the most responsive 12. Most scores lie between these extremes. Unpublished data, Stanford Laboratory.

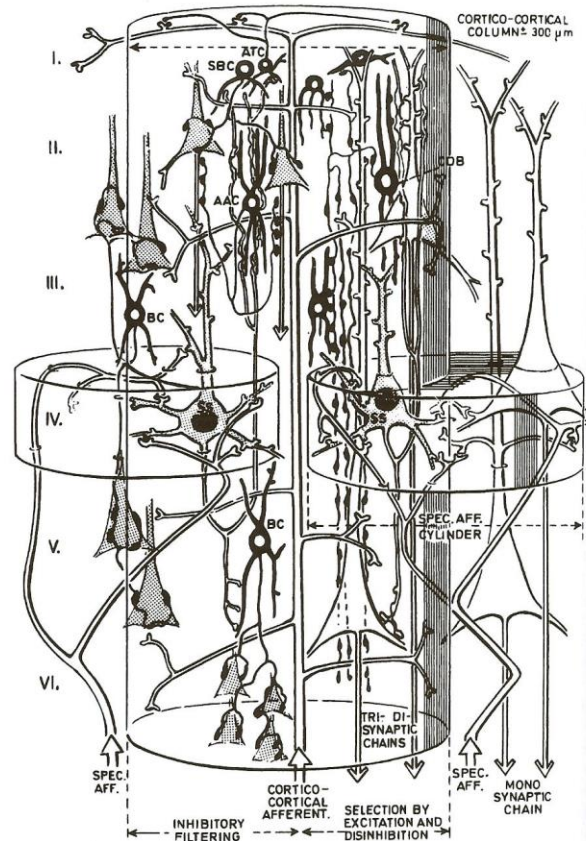


# Brain Wave State Diagram

Brain Wave Frequency/Level		Mental Characteristics	Physical Characteristics	Synchronization Effect
Gamma 30+ cps	120	Flow State	Energized, Fluid, Graceful	Re-synchronized neural functions.
	115	One-pointed concentration	Feelings of Enjoyment, Increased Stamina	
	110	Focused	Increased Coordination	
	105	Increasing Focus	Increased Fluidity	
Beta (13 – 30 cycles per second)	100	Wide Awake State	Extreme Tension, Uptight	Alpha-blocking and De-synchronization of neural functions. The brain is processing 'external' information, and each functional sub-unit [Broca's Area, Wernicke's Area, Visual Cortex, Motor Cortex, etc.] is operating independently.
	95	Excitement, Frustration	High Metabolic Behavior	
	90	Aware of all senses	Hands Moist and Clammy	
	85	Very Alert	Accelerated Work Ability	
	80	Actively Aware	Hyperactive	
	75	Active Thought Patterns	High Degree of Stamina	
	70	Comfortably Alert	Comfortable, Restful State	
	65	Consciously Aware	Good Observation State	
	60	Normal Thought Patterns	Physically At Rest	
	55	Easy Thoughts	Beginning to Relax	
	50	Less Active Thoughts	Increased Composure	
Alpha (8-12 cps)	45	Pre-Drowsiness	Releasing All Body Feelings	Synchronized neural functions. All CNS neurons are firing at the same rate in the same rhythm.
	40	Increased Susceptibility	Passive Awareness	
	35	Passive Awareness	Numb, Quiet	
	30	Total Sensory Withdrawal	Deep Relaxation	
	25	Low Alpha State	Complete Passivity	
Theta (4-7 cps)	20	Drowsiness	Unaware	De-synchronization as various functional subunits go offline. Hippocampus very active – PGO spikes.
	15	Beginning Unconsciousness	Unaware	
	10	Unconsciousness	Unconscious	
Delta (.05-3.0 cps)	5	Deep Sleep State	Deep Sleep State	Non-synchronization as neo-cortex rests during slow-wave sleep cycle.
	0	Baseline	Baseline	

# Cortical Module

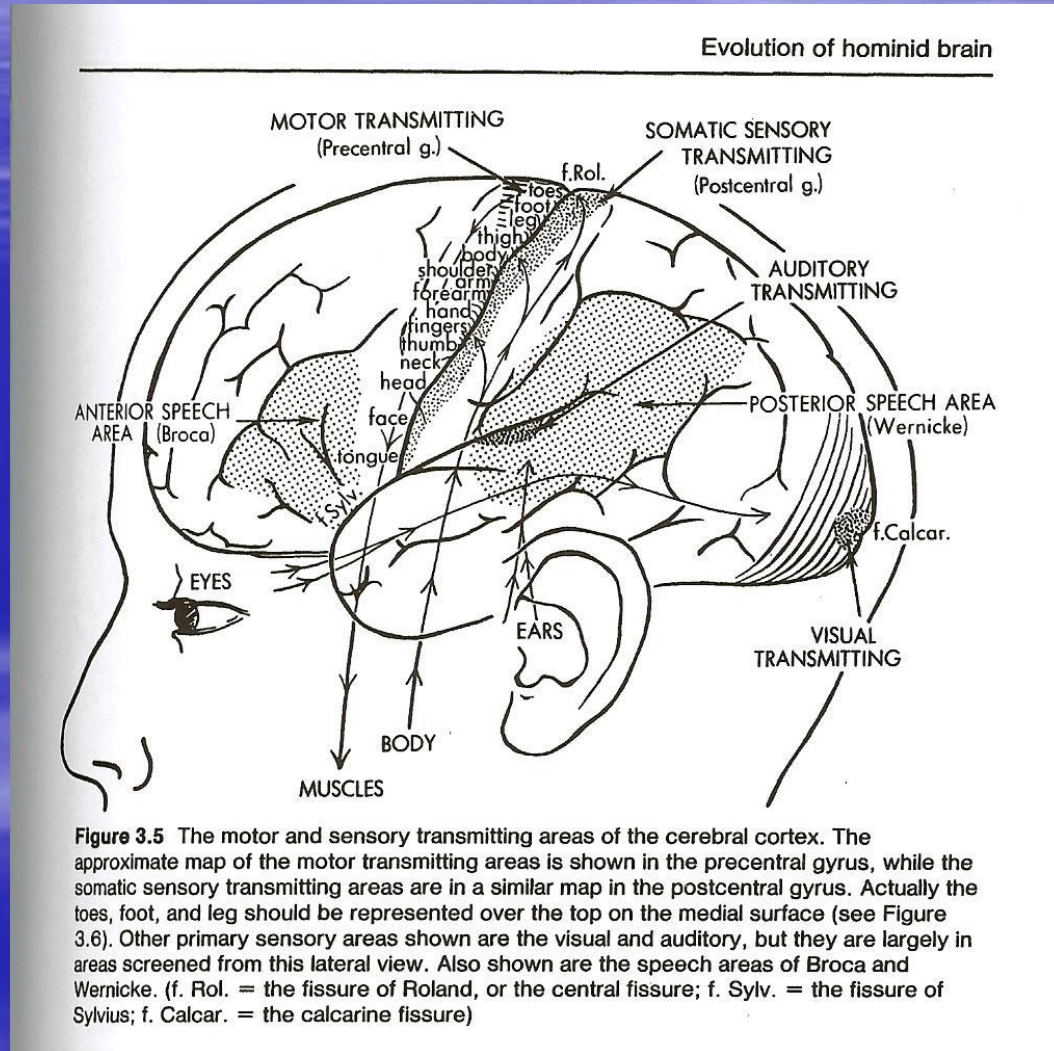
Evolution of the brain: creation of the self.



**Figure 3.4** Neuron connectivity in a corticocortical column or module (see Figure 9.4), the vertical cylindrical space of about 300 μm in the centre. The module is sharing part of its space with two flat disks in lamina IV, in which specific afferents (SPEC. AFF.) arborize. The corticocortical afferent (indicated at bottom) terminates all over the corticocortical module, though with different densities of terminals. In lamina I, the tangential spread of the

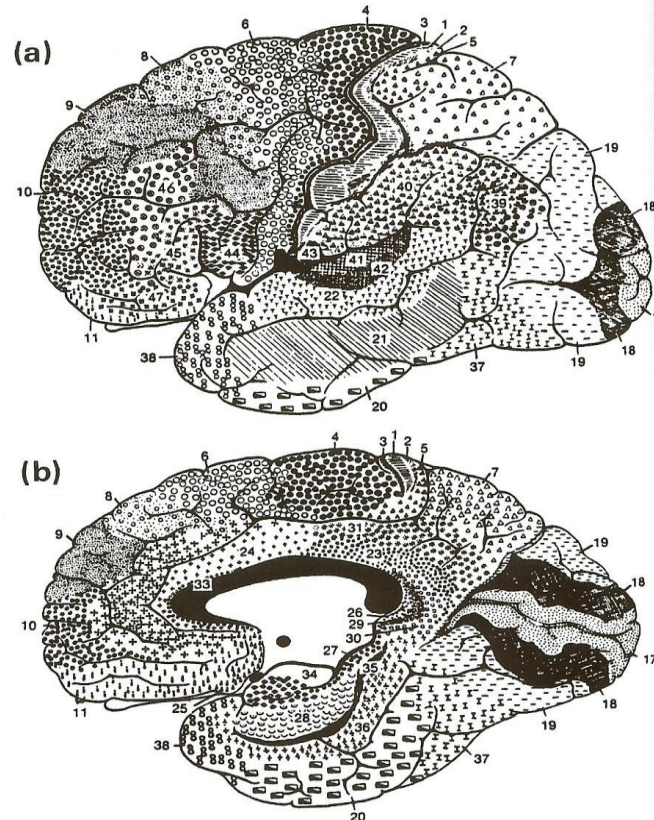


# Motor-Sensory Areas



# Brodmann Areas

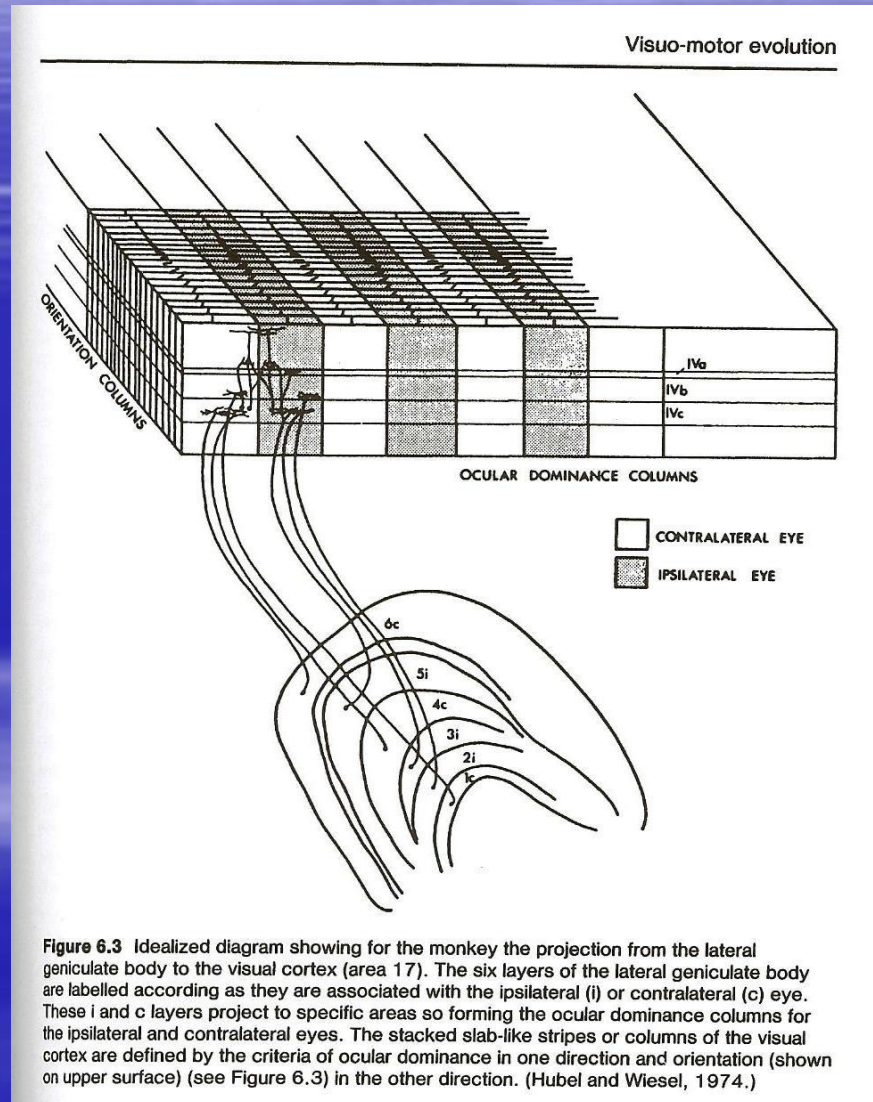
Evolution of the brain: creation of the self



**Figure 4.4** Brodmann's cytoarchitectural map of the human brain. The various areas are labelled with different symbols and their number indicated by figures. (a) Lateral view of left hemisphere. (b) Medial view of right hemisphere. (Brodmann, 1909, 1912.)



# Visual Cortex Mapping



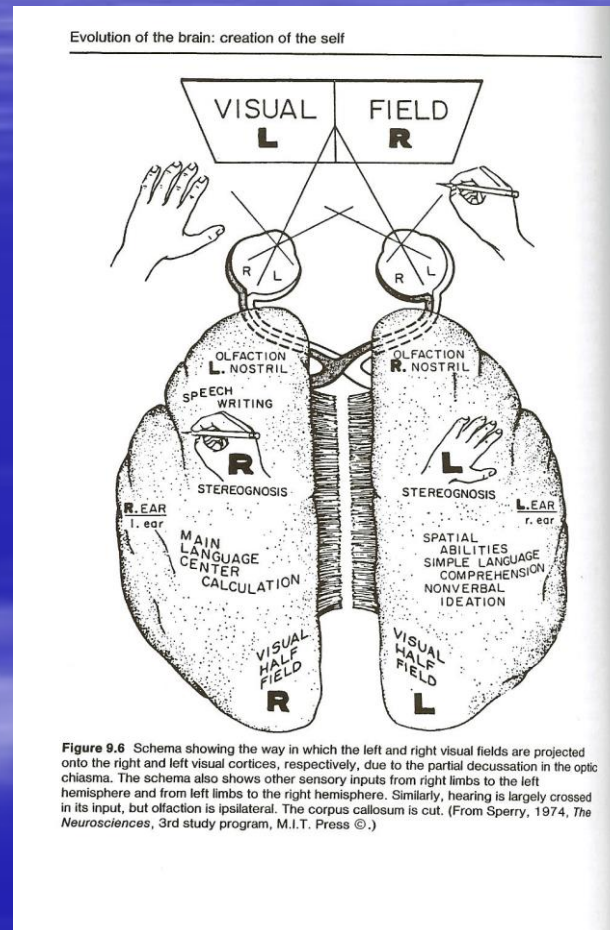
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graph TD
    L((L)) --> SC[SC]
    S((S)) --> SC
    SC --> LMA((LMA))
    SC --> MT((MT))
    SC --> LFS((LFS))
    SC --> SST((SST))
    LMA --> MT
    MT --> LFS
    LFS --> FC[FC]
    FC --> AC[AC]
    AC <--> SSC[SSC]
    SSC --> SST
    SST --> SC
    subgraph ShadedArea [ ]
        LMA
        MT
        LFS
    end
    style ShadedArea fill:#ccc,stroke:#333,stroke-width:1px

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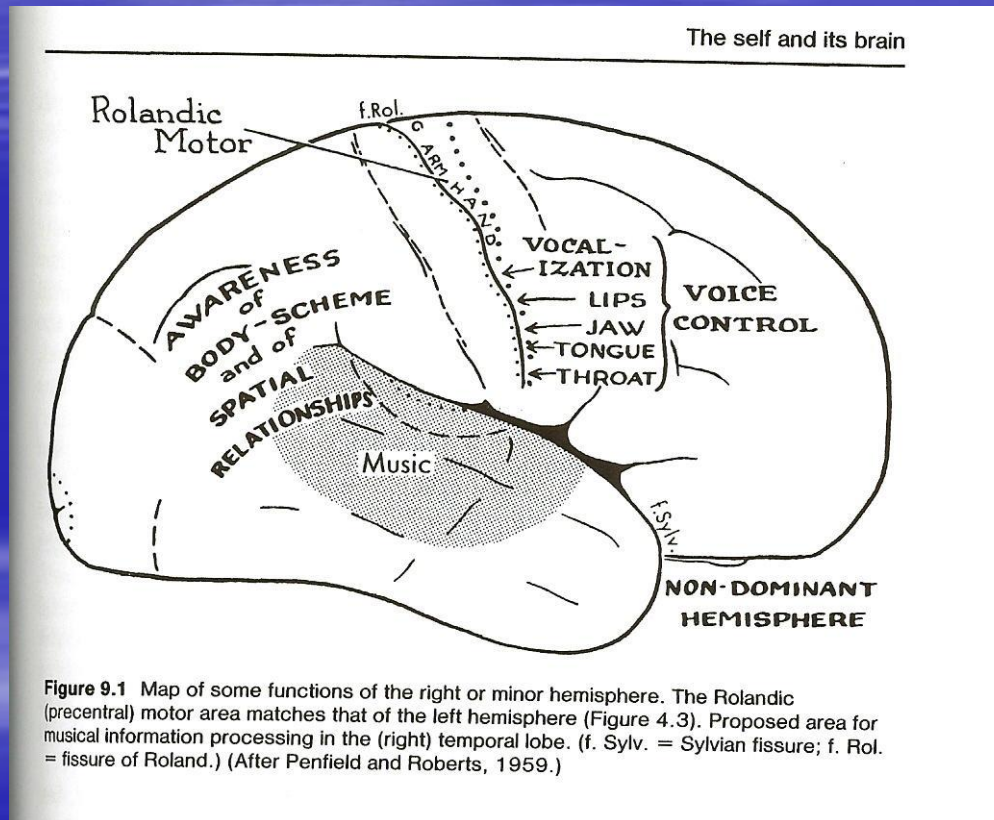
**Figure 13 Schematic diagram of the major relationships among structures in the central nervous system that are related to pain.** On the right: thalamic and neocortical structures subserving discriminative capacity. On the left: reticular and limbic systems subserving motivational-affective functions. Ascending pathways from the spinal cord (SC) are: (1) the dorsal column-lemniscal and dorsolateral tracts (right ascending arrow) projecting to the somatosensory thalamus (SST) and cortex (SSC), and (2) the anterolateral pathway (left ascending arrow) to the somatosensory thalamus via the neospinothalamic tract, and to the reticular formation (stippled area), the limbic midbrain area (LMA) and medial thalamus (MT) via the paramedial ascending system. Descending pathways to spinal cord originate in somatosensory and associated cortical areas (AC) and in the reticular formation. Polysynaptic and reciprocal relationships in limbic and reticular systems are indicated. Other abbreviations: FC – frontal cortex; LFS – limbic forebrain structures (hippocampus, septum, amygdala, and associated cortex); H – hypothalamus.  
(from Melzack and Casey, 1968)

# Hemispheric Lateralization





# Non-Dominant Hemisphere



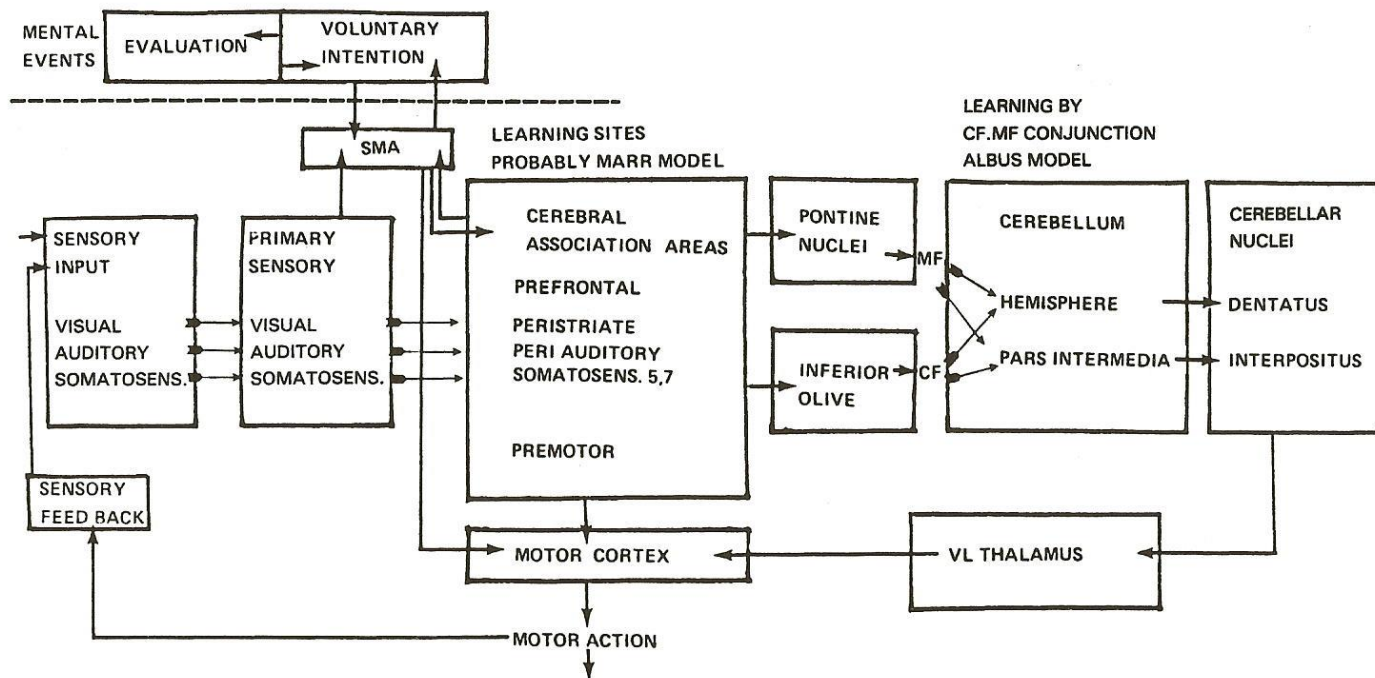


# Hemispheric Specialization

DOMINANT HEMISPHERE	MINOR HEMISPHERE
Liaison to self-consciousness	Liaison to consciousness
Verbal	Almost non-verbal
Linguistic description	Musical
Ideational	Pictorial and pattern sense
Conceptual similarities	Visual similarities
Analysis over time	Synthesis over time
Analysis of detail	Holistic — Images
Arithmetical and computer-like	Geometrical and spatial

**Figure 9.7** Various specific performances of the dominant and minor hemispheres as suggested by the new conceptual developments of Levy-Agresti and Sperry (1988) and Levy (1978). There are some additions to their original list.

# Learning Flowchart



**Figure 7.13** Diagrammatic representation of the proposed mental and neural events in learning a skilled movement. Mental events for human motor learning are shown above the dashed line that separates them from the neural events. The role of mental events in animal motor learning is left undefined. The arrows indicate directions of main pathways of action. See the text for a full description. All pathways are excitatory except for the inhibitory action of the cerebellum on the cerebellar nuclei.

# Placebo Response

## BOX 1 Comparing Efficiency of Placebo and Analgesics

Illustration of Calculation of Index of Drug Efficiency for Evaluating Placebo Efficiency Compared to Analgesic Drugs

*Index of analgesic drug efficiency:*

$$\frac{\text{Reduction in pain with unknown drug}}{\text{Reduction in pain with known analgesic (typically morphine)}}$$

*Pain criterion:*

Reduction in pain by 50% of initial level over drug level.  
or  
change in pain of 50% on rating scale (typically 10- or 5-point)

*Index of placebo efficiency for morphine:*  
(averaged across six double-blind non-crossover-design studies)

$$\frac{\text{Reduction in pain with placebo}}{\text{Reduction in pain with morphine}} = 56\%$$

Index of Placebo Efficiency Comparing Placebo with Morphine, Aspirin, Darvon, and Zomax (Derived from Available Single-Trial Double-Blind Published Studies)

Number of double-blind studies	Placebo efficiency for	%
6	Morphine	56
9	Aspirin	54
2	Darvon	54
2	Codeine	56
3	Zomax	55

Used by permission from Evans (1985).

# Autonomic Nervous System

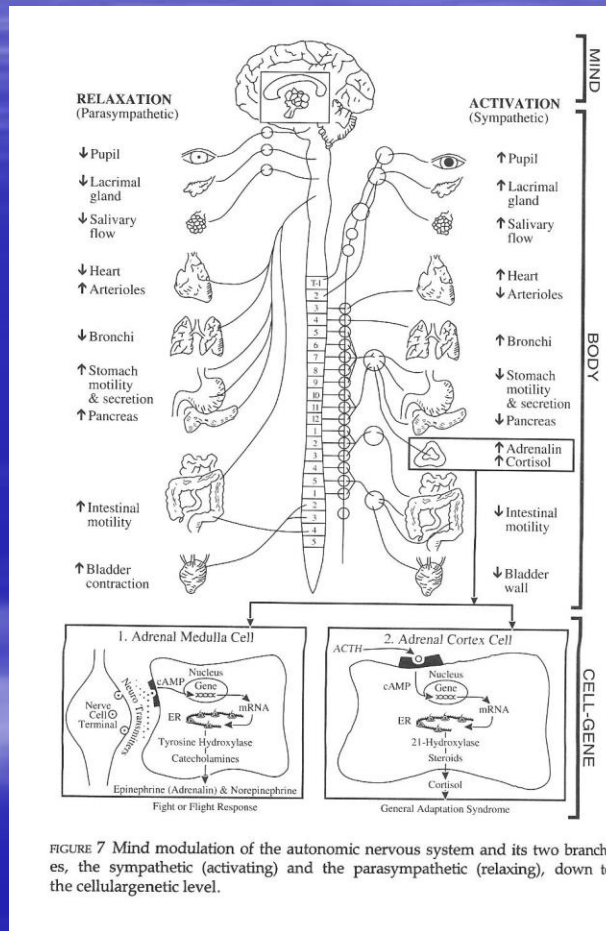


FIGURE 7 Mind modulation of the autonomic nervous system and its two branches, the sympathetic (activating) and the parasympathetic (relaxing), down to the cellular/genetic level.



# Cybernetic Loop

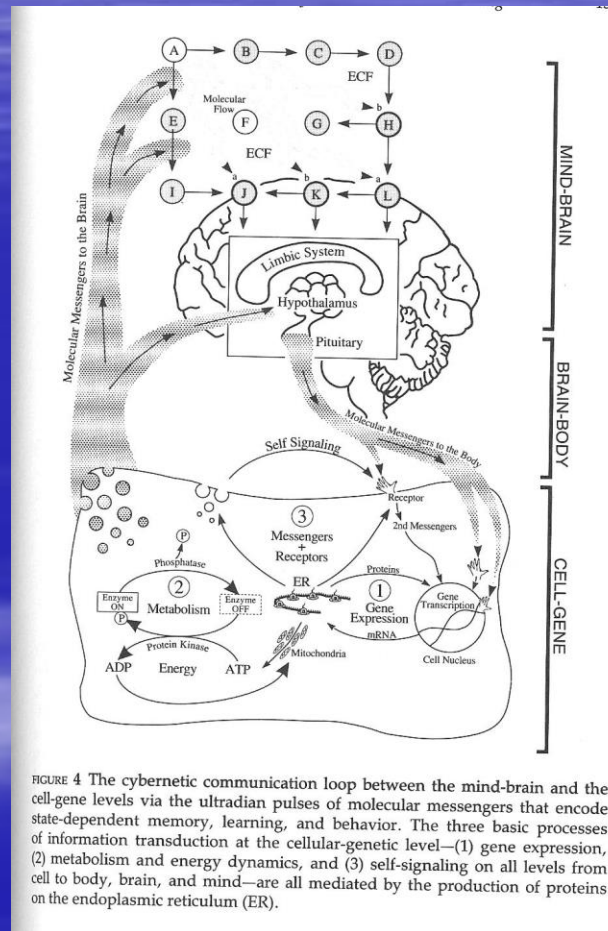
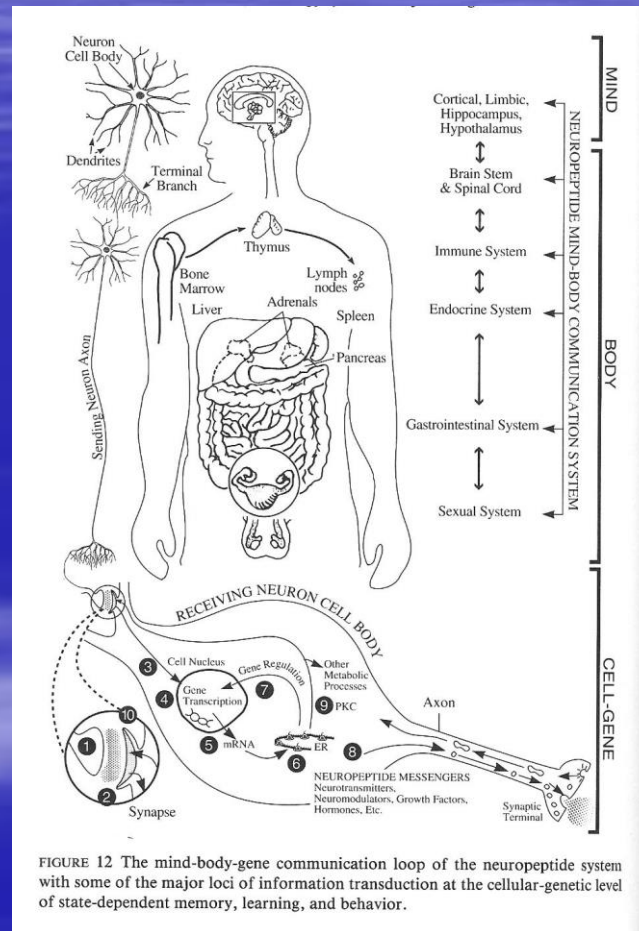
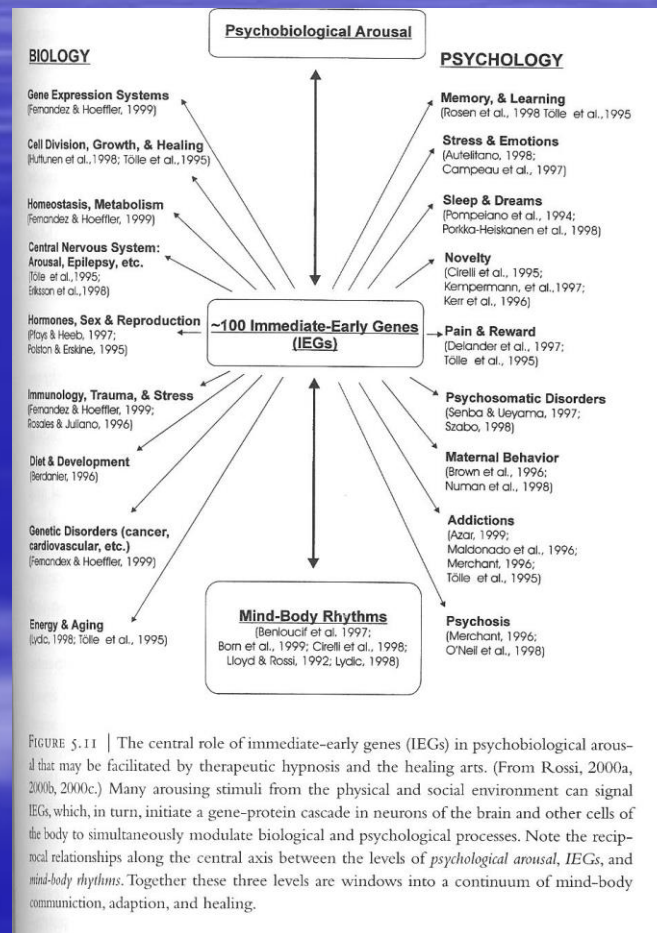


FIGURE 4 The cybernetic communication loop between the mind-brain and the cell-gene levels via the ultradian pulses of molecular messengers that encode state-dependent memory, learning, and behavior. The three basic processes of information transduction at the cellular-genetic level—(1) gene expression, (2) metabolism and energy dynamics, and (3) self-signaling on all levels from cell to body, brain, and mind—are all mediated by the production of proteins on the endoplasmic reticulum (ER).

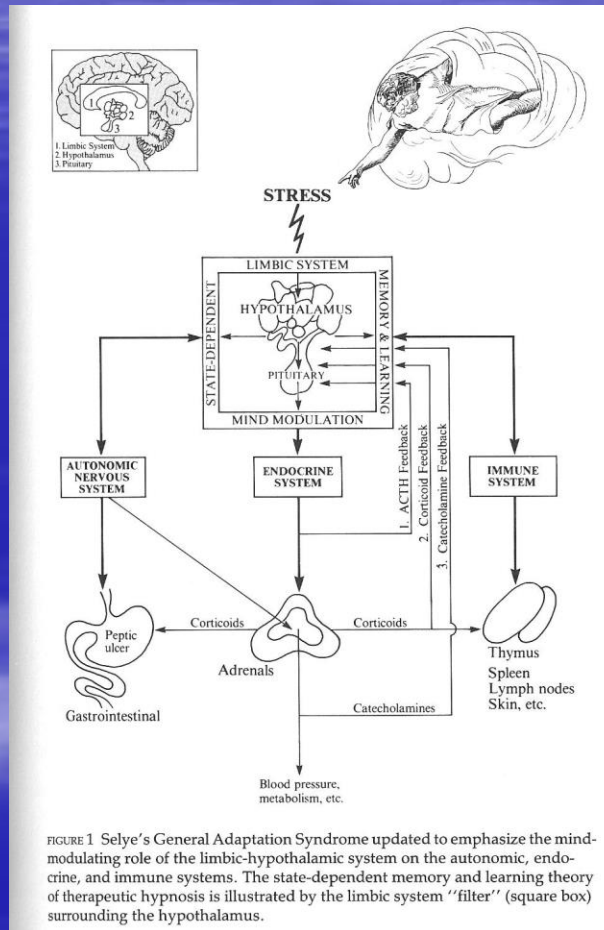
# Neuropeptide Loop



# Immediate-Early Gene Expression

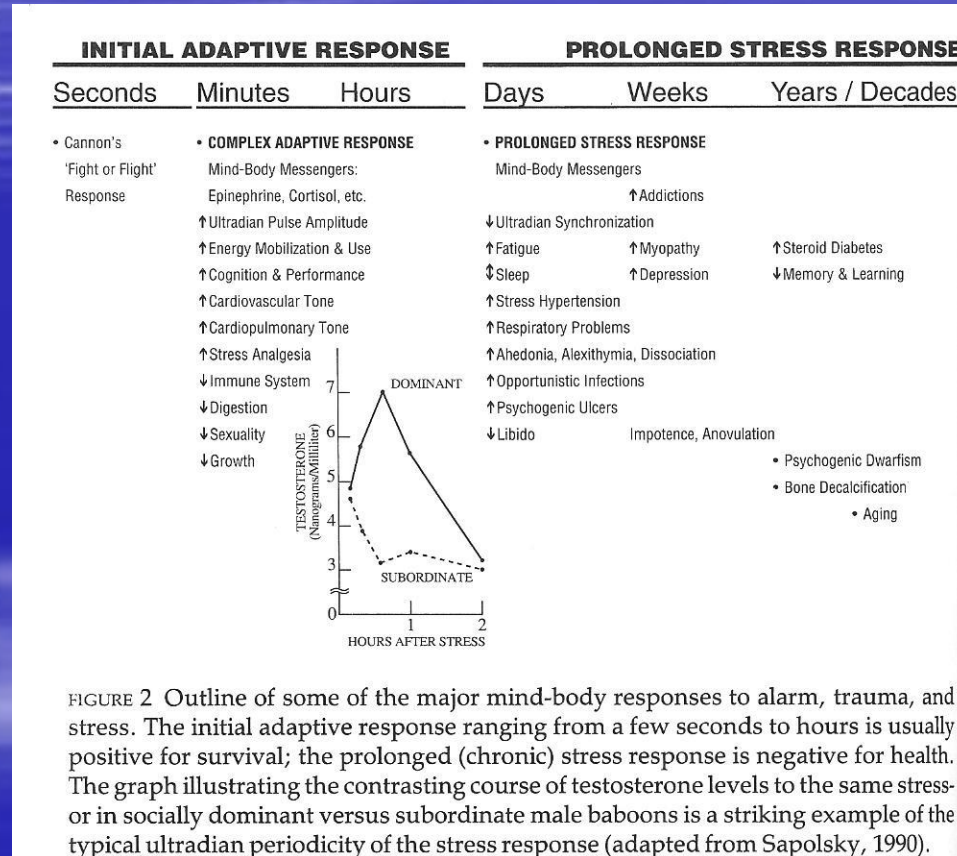


# General Adaptation Syndrome





# Stress Response



# Ultradian Rhythms

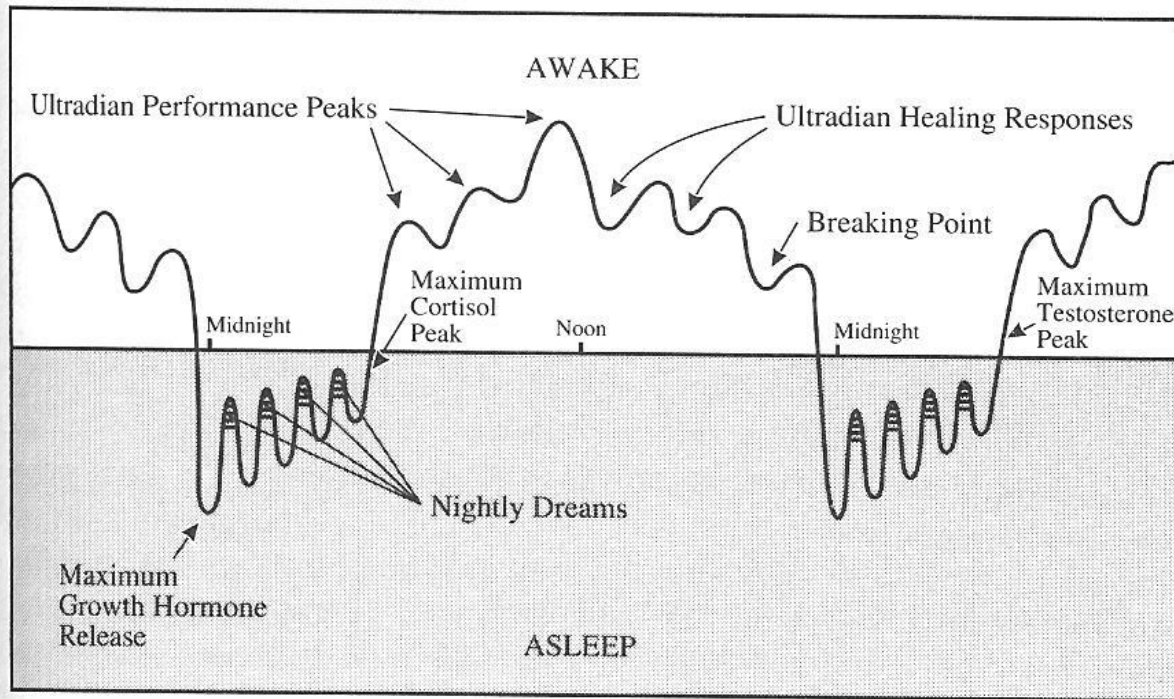


FIGURE 9 The wave nature of consciousness and being. An idealized illustration of the alternating character of our natural ultradian performance peaks, healing responses, and dreams.



The diagram illustrates the IL-2 signaling pathway. IL-2 binds to the IL-2 receptor complex (IL-2Rα, IL-2Rβ, IL-2Rγ), which activates JAK1 and JAK3. JAK1 and JAK3 phosphorylate Lck and SYK, which then activate STAT5. The Ras/MAPK pathway is also activated via Shc, Grb2, Sos-1, Ras, Raf1, MEK, and ERK. These pathways lead to the activation of transcription factors like c-Fos, c-Jun, and AP-1, which bind to DNA to regulate gene expression.

# Short and Long Term Memory

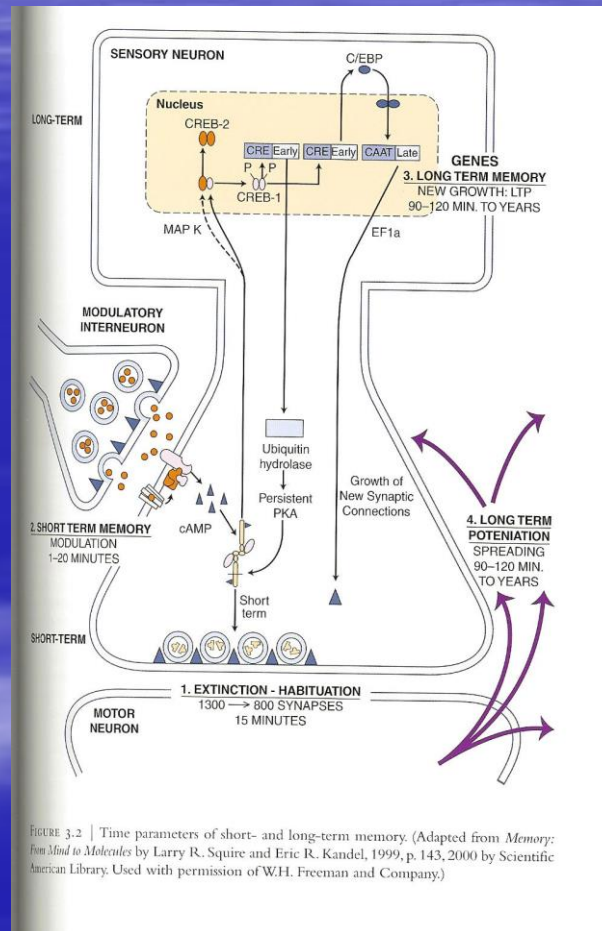


FIGURE 3.2 | Time parameters of short- and long-term memory. (Adapted from *Memory: From Mind to Molecules* by Larry R. Squire and Eric R. Kandel, 1999, p. 143, 2000 by Scientific American Library. Used with permission of W.H. Freeman and Company.)



# PET Scan of Gene Expression

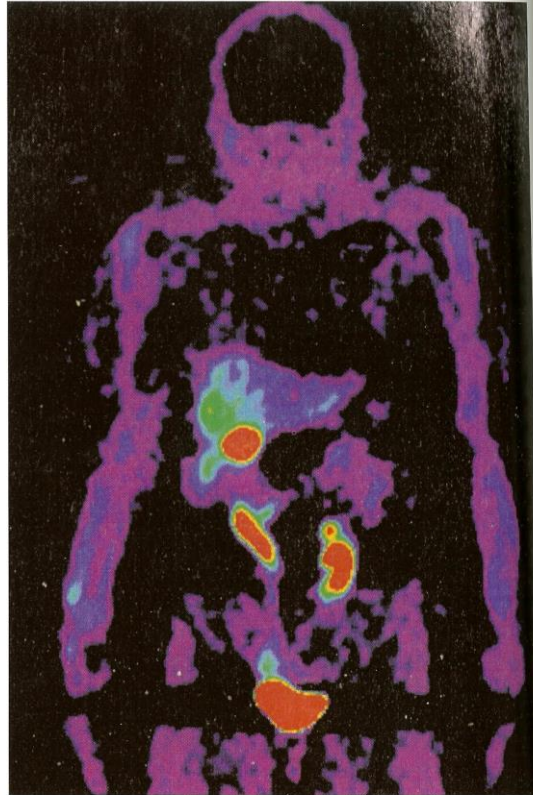


FIGURE 5.9 | A color-coded positron emission tomography (PET) scan of the human body illustrating areas of heightened gene expression in the liver, intestines, and bladder. (With permission from Yaghoubi et al., 2001.)

# Discussion Period - Questions and Answers

# Section 11

Ideo-Dynamics:  
Behavioral Kinesiology  
Demonstration



# 4-MAT Frame for Section 10

- What Frame: This section presents information on:
  - Behavioral Kinesiology and its relation to ideomotor effects
  - A demonstration of BK

# Review of Ideo- Dynamic Processes

# Behavioral Kinesiology Demonstration



# Discussion Period - Questions and Answers

# Section 12

## Hypnosis in the Treatment of Pain

# 4-MAT Frame for Section 10

- What Frame: This section presents information on:
  - Hypnosis and its success in treating pain
    - Experimental Findings
    - Historical successes
    - Contemporary successes
    - Cognitive Restructuring Model of Hypnoanalgesia



# Experimental Hypnotic Analgesia

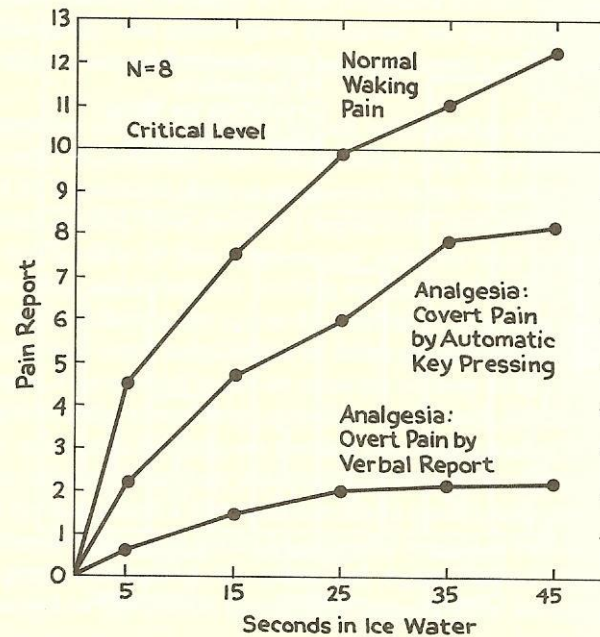
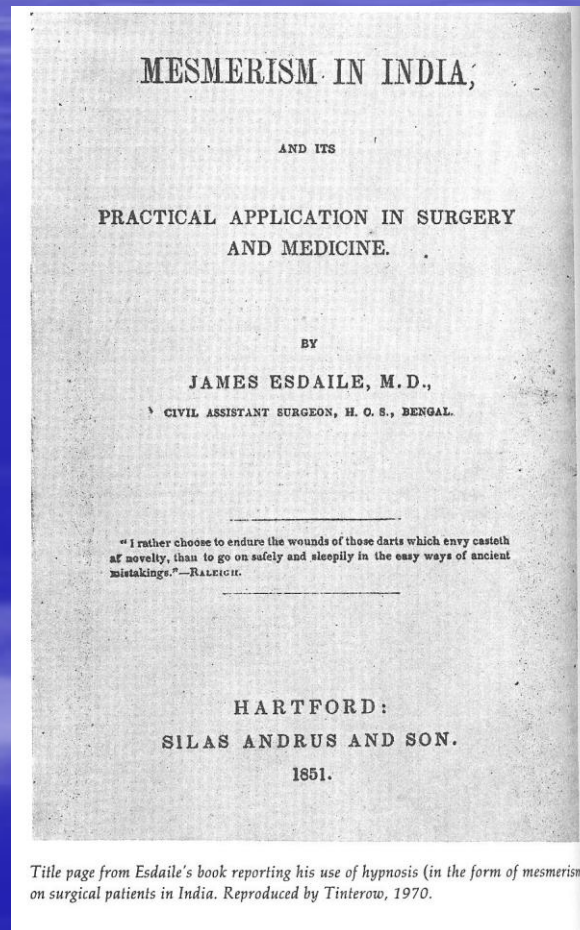


Figure 13. Normal waking pain, and overt and covert pain in hypnotic analgesia.

Results are for the eight most successful subjects from those twenty whose means are reported in Figure 12. The covert reports were obtained by automatic key-pressing.

# Dr. James Esdaile



# Dental Procedures

**Table 3.** Some Case Reports of Dental Procedures with Hypnosis as the Sole Anesthetic.

<i>Source (in chronological order)</i>	<i>Patients</i>	<i>Nature of Procedure</i>
Traiger (1952)	(a) 4-year-old (b) 9-year-old	(a) Pulpectomy and pulpotomy (b) Four pulpotomies during 3 hrs.
Crasilneck, McCranie, and Jenkins (1956)	Woman patient	Complex procedures on 5 occasions, each lasting 2 hrs.
Lucas, Finkelman, and Tocantins (1962)	Hemophiliacs (4 cases)	Extractions
McCay (1963)	Male physician under self-hypnosis	Extraction
Secter (1964)	Patient in spontaneous trance	Extraction of two mandibular premolars
Petrov, Traikov, and Kalendgiev (1964)	(a) Woman, age 35 (b) Woman, age 20 (c) Woman, age 25	Extractions, including one difficult case lasting 1½ hours
Owens (1970)	(a) Woman, age 35, with multiple sclerosis (b) Woman, age 41	(a) Extraction of 2 abscessed teeth (b) Extraction of 2 abscessed teeth
Radin (1972)	(c) Male, age 49 Adult male; chemo-anesthesia contra-indicated	(c) Periodontal curettage Extensive extractions on repeated occasions, including suturing
Weyandt (1972)	Male, age 65	Seven teeth extracted



# Surgical Procedures

**Table 2.** Some Operations in which Hypnotic Analgesia or Anesthesia or Anesthesia was used with no Chemical Analgesics or Anesthetics, 1955-1974.

<i>Type of Operation</i>	<i>Reference</i>	<i>Type of Operation</i>	<i>Reference</i>
<i>Abdominal Surgery</i>		<i>Genitourinary</i>	
Appendectomy	Tinterow (1960)	<i>continued</i>	
Caesarean section	Kroger and DeLee (1957)	Vaginal hysterectomy	Tinterow (1960)*
	Taughner (1958)	Circumcision	Chong (1964)
	Tinterow (1960)*	where phimosis present	
Gastrostomy	Bonilla, Quigley, and Bowers (1961)	Prostate resection	Schwarcz (1965)
		Transurethral resection	Bowen (1973)
<i>Breast Surgery</i>		Oophorectomy	Bartlett (1971)
Mammoplasty	Mason (1955)	<i>Hemorrhoidectomy</i>	Tinterow (1960)*
Breast tumor excision	Kroger (1963)	<i>Nerve Restoration</i>	
Breast tissue excision	Van Dyke (1970)	Facial nerve repair	Crasilneck and Jenkins (1958)
<i>Burns</i>		<i>Thyroidectomy</i>	
Skin grafting, debridement, etc.	Crasilneck, McCranie, and Jenkins (1956)		Kroger (1959)
	Tinterow (1960)		Chong (1964)
	Finer and Nylén (1961)		Patton (1969)
<i>Cardiac Surgery</i>	Marmar (1959a)	<i>Venous Surgery</i>	
	Tinterow (1960)	Ligation and stripping	Tinterow (1960)
<i>Cataract Excision</i>	Ruiz and Fernandez (1960)	<i>Miscellaneous</i>	
<i>Fractures and Dislocations</i>	Goldie (1956)	Removal of tack from child's nose	Bernstein (1965b)
	Bernstein (1965)	Repair of lacerated chin in child	Bernstein (1965b)
<i>Genitourinary</i>		Removal of fat mass from arm	Scott (1973)
Cervical radium implantation	Crasilneck and Jenkins (1958)		
Curettage for endometritis	Taughner (1958)		

\*Some nonanalgesic medication used during preoperative or surgical procedures.



# Heart Rate

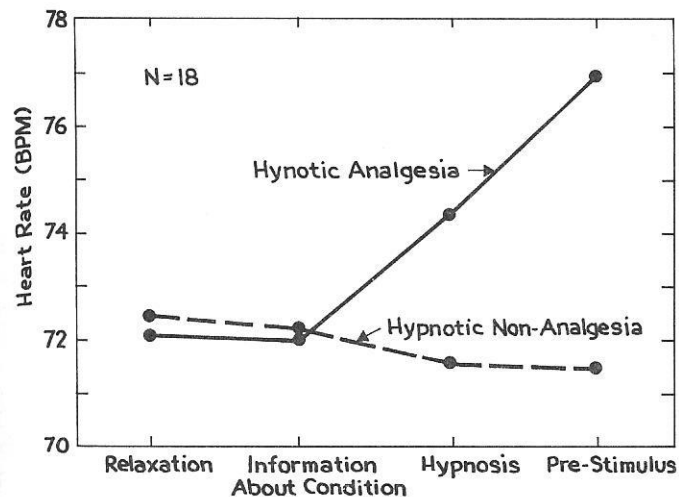
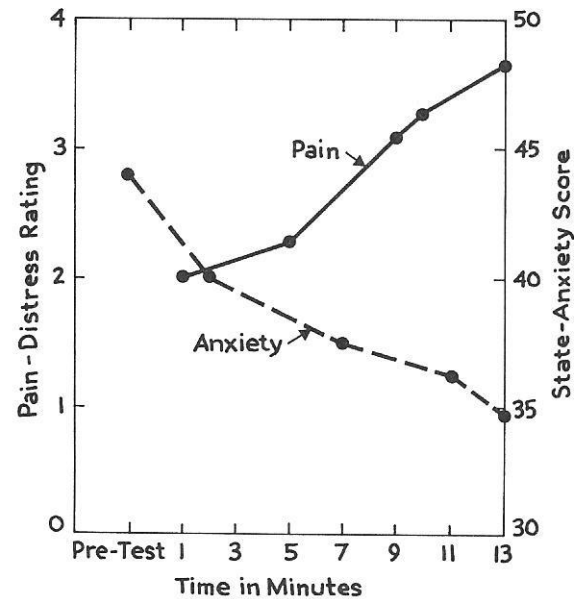


Figure 10. Anticipatory heart rate changes prior to immersion of hand in ice water under hypnosis, with and without analgesia suggestions. The experimental arrangements were those described earlier. Note that the greatest changes occurred in hypnotic analgesia, when subjects anticipate feeling no pain or greatly reduced pain. After Hilgard, Macdonald, Marshall, and Morgan (1974).

# Pain-Anxiety Curve



**Figure 9.** Separateness of pain and anxiety following a tranquilizer. Tourniquet pain increases over time, while state-anxiety is decreasing. Modified from Chapman and Feather (1973), combining data from their Figs. 1 and 2.

# Susceptibility

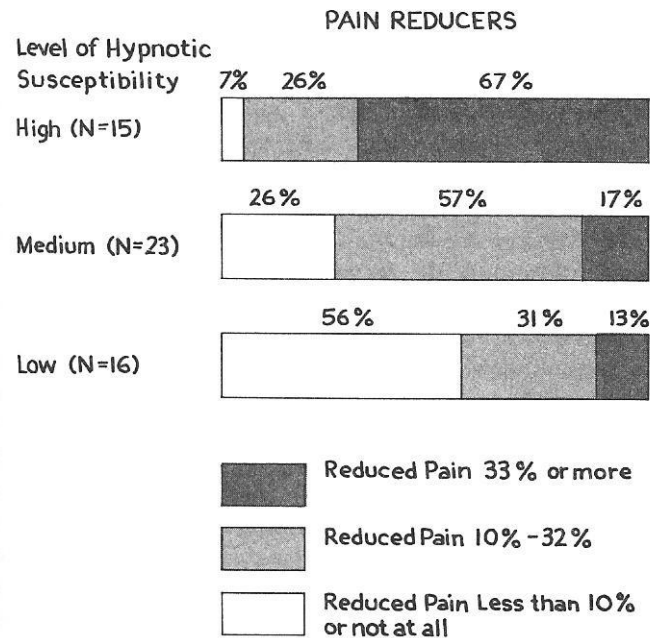


Figure 7. Reduction of pain through hypnotically suggested analgesia as related to susceptibility to hypnosis. The subjects were fifty-four university subjects whose prior experience of hypnosis was limited to a standard test of hypnotic responsiveness following a standardized induction procedure. From Hilgard and Morgan (1976).

# Cognitive Restructuring Model

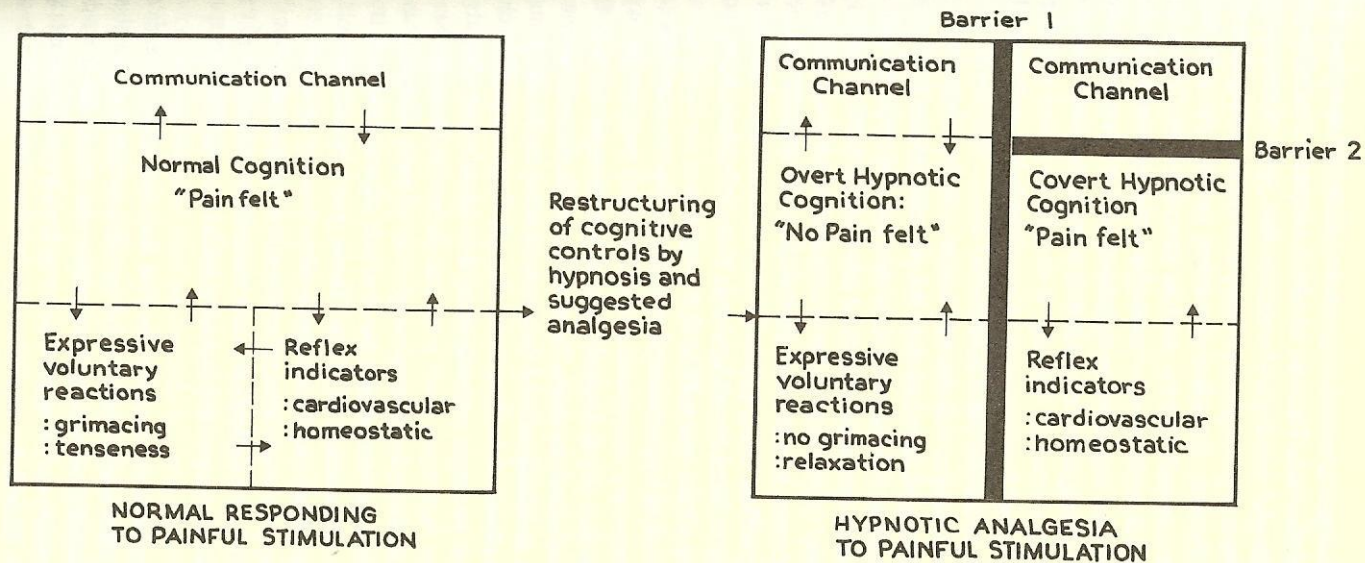


Figure 15. Restructuring of cognitive controls in hypnotic analgesia.



# Discussion Period - Questions and Answers

# Section 13

## Introduction to the Trance Experience

# Discussion and Review